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THE ELECTROENCEPHALOGRAMS OF PSYCHOTIC PATIENTS.*, 1, 2

By PAULINE A. DAVIS, M. A., AND HALLOWELL DAVIS, M. D.,
Department of Physiology, Harvard Medical School.

The electroencephalogram (EEG) is a record of the physiological activity of the cortex. Two years ago we made the statement (1) that different persons show different patterns of cortical activity, but that each person reproduces his own characteristics in successive records taken under standard conditions. Furthermore, we pointed out the remarkable similarity of pattern in 14 pairs of identical twins. Our original statement was based on a study of 100 normal adults. We now have 500 normals in our series and their records show individuality of pattern and consistent reproducibility over a period of three years.

The original purpose of the present study was twofold: first, to discover whether EEG's of psychotics differ from the normal patterns, and secondly, to determine whether the brain-wave pattern of a psychotic patient changes as he shifts, let us say, from manic to depressed condition.

The EEG's of 180 unselected patients at McLean Hospital in Waverly, Mass., have been studied since October, 1936.[3] More recently 52 chronic schizophrenics at the Metropolitan State Hospital have been added to the series and their records repeated after an interval of nearly six months. The technic of recording has been kept standardized throughout. Standard conditions imply that the person be lying on a bed in a comfortable position with eyes closed and at ease in relation to his surroundings. An ink-

* Read at the ninety-fourth annual meeting of The American Psychiatric Association, San Francisco, Calif., June 6-10, 1938.

1 Aided by a grant from the Josiah Macy, Jr. Foundation.

2 The complete freedom of research facilities and unstinted coöperation of members of the staff at the McLean Hospital for the last two years, and at the Metropolitan State Hospital this year, have made this research possible.

3 The McLean Hospital cases include patients diagnosed as manic-depressive, schizophrenic, psychoneurotic, alcoholic, psychopathic personality, involutional psychosis, and psychosis with associated organic disturbances including a few epilepsies.

writing oscillograph and condenser-coupled amplifiers built by A. M. Grass of our laboratory have been used throughout.[4]

Psychiatrists may be interested to know that the EEG has already proved to be of practical service in revealing unsuspected neurological conditions.

We have previously published a figure (3) which shows the extremes of pattern found among normal individuals. The most prominent and easily measurable feature is a rhythm of about 10 waves a second which may persist 100 per cent of the time in some records, part of the time in others, or it may even be absent all of the time in some people. Smaller waves at faster frequencies appear normally in almost all records.

Fig. 1 shows how the EEG's of schizophrenic and also of manic-depressive patients, both manic and depressed, fit into the normal range of patterns. Several of them show 10/sec. waves nearly all the time; another has none of them. These individuals have been recorded many times and their patterns have not changed. The manic phase cannot be distinguished from the depressed by the electroencephalogram.

We considered specifically the feature which is most prominent in the EEG of the normals, the 10/sec. rhythm. We measured the percentage of time this was present in a person's occipital record (the "alpha index"). In most normal subjects under standard conditions, the percentage does not vary on successive tests more than 10 points on either side of the average. Separating schizophrenic and manic-depressive patients there is no significant difference in the distribution of the alpha index between these two groups. We plotted the entire hospital group in relation to our normals, although, unlike most normals, many psychotics vary greatly from test to test in their alpha indices. Fig. 2 shows that there is an almost inverse trend for this single common element of the records when contrasting normals and psychotics. Such a single element may serve to show a statistical difference between the two groups, but we believe that this or any other single element is far less significant than a series of judgments based on each record as a whole.

[4] The amplifiers and writers are duplicates of those used by Drs. Gibbs and Lennox at the Boston City Hospital. Details, such as the type of electrode employed and their placement, have been described elsewhere (2).

The importance of this statement lies in the fact that although the records of many psychotics cannot be distinguished from the normal, we nevertheless find in a larger proportion of the records of patients in a mental hospital, variations which we have come to recognize as definitely abnormal (*cf.* also 5, 11 and 6). The EEG's of 100 normal and 100 mental patients were judged on the basis of abnormal qualities which will be illustrated later in detail.

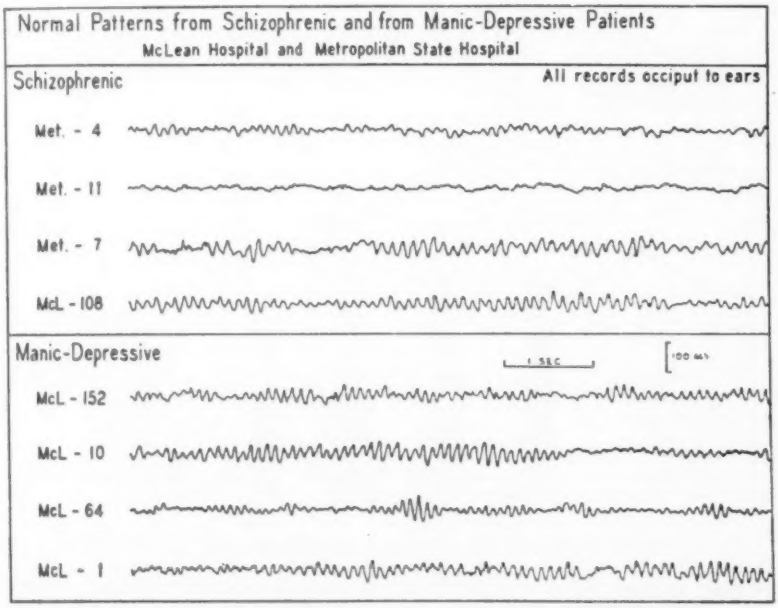

FIG. 1.—Occipital EEG's of eight McLean Hospital or Metropolitan State Hospital patients with clinical diagnosis of schizophrenia or manic-depressive psychosis. All of these records lie within the normal range of variation. These EEG's and all others illustrated in this paper, unless otherwise specified, were recorded with one electrode attached to the scalp in the midline in the region indicated and two reference electrodes connected in parallel attached to the lobes of the ears. Patients were lying quietly on a bed with eyes closed. Upward deflection indicates scalp becoming electrically more negative relative to the ears.

In order to judge a pattern a minimum of 15 minutes and preferably half an hour of record should be taken. We have recorded from the occiput, the vertex, and the frontal region of the head and have based our judgments on the relationships between them as well as the quality of each record separately. Fig. 3 shows the

distribution of normals and of psychotics, rated on a scale of 1 to 5. One means normal, 2 slightly irregular, 3 borderline, 4 highly irregular, and 5 abnormal. (The detailed definitions of our ratings

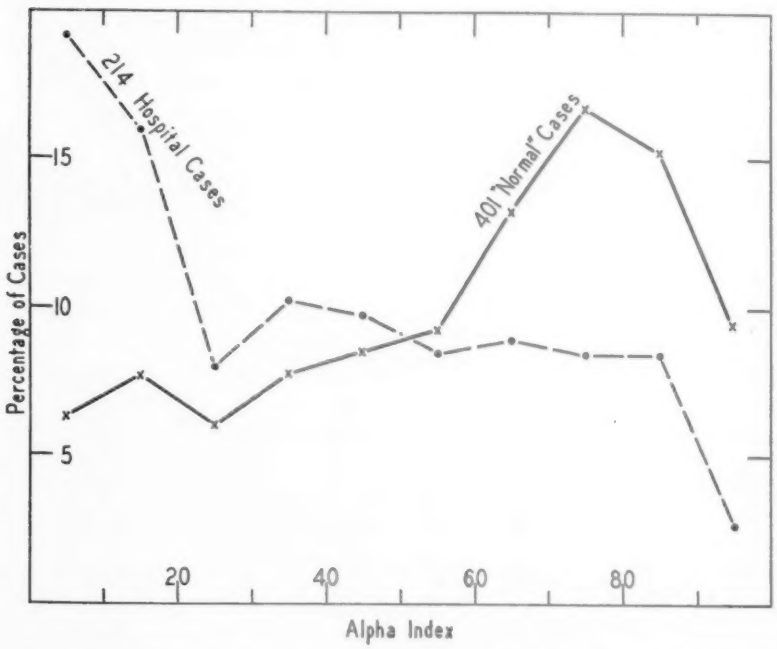

FIG. 2.—Distribution of 401 "normal" individuals and of 214 patients in mental hospitals (McLean Hospital and Metropolitan State Hospital) according to their alpha indices. These alpha indices were measured on the first record of each person. The alpha index is the percentage of time that the alpha rhythm (8.5 to 13 per second) appears in the occipital record of a patient under standard conditions with eyes closed. Only waves which are in approximately regular sequences of at least three waves, all at least 7 microvolts in amplitude, are counted. Two or more samples of at least thirty seconds each are measured. Identification of alpha waves is facilitated by placing a broadly tuned electrical filter in the amplifier circuit (2).

The patients in this series were diagnosed as follows:

Schizophrenia (all types) 93,
Manic-depressive psychosis 64,
Psychosis with cerebral arteriosclerosis 9,
Epilepsy 7,
Miscellaneous (see footnote 3, p. 1007) 41.

1 to 5 are somewhat technical and cannot be adequately summarized in this condensed report. They will be dealt with in a separate paper.) The large proportion of abnormalities in the

records of the psychotic group is obvious in Fig. 3. Although the psychotic individual cannot be recognized by his EEG, nevertheless as a group the psychotics have a significantly larger percentage of abnormalities in their EEG's than do normals.

Abnormal qualities appear in the EEG when physiological or pathological changes take place in the brain. If a normal person

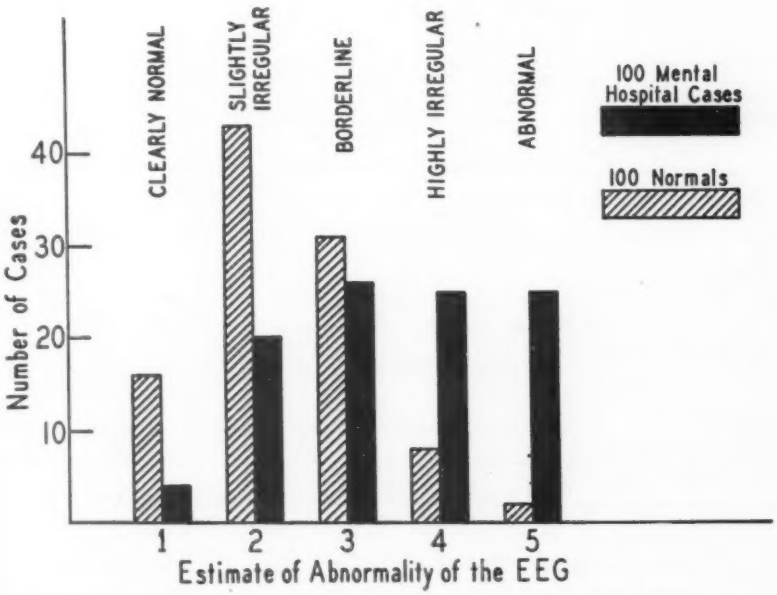

FIG. 3.—Distribution of 100 consecutive normal individuals and of 100 consecutive patients in mental hospitals, according to the estimate of the degree of abnormality of their EEG's (see text). The patients include 52 with chronic schizophrenia (Metropolitan State Hospital) and 48 with various other diagnoses (McLean Hospital).

breathes an inadequate supply of oxygen for a long enough time to affect the mental state, the EEG is altered (7), as shown in Fig. 4, but the record resumes its normal pattern when room air is breathed again. The mental state also returns to normal.

The profound changes of the EEG seen in sleep (8, 2) and other forms of unconsciousness such as coma and anæsthesia (9, 10, and 11) are becoming more and more familiar. Fig. 5 shows the EEG's from the motor area and from the occiput of a normal person awake and asleep. This is to illustrate that in sleep

the EEG of the normal brain can and does undergo profound changes. For this reason one cannot be too careful to run records under standard conditions, which means that the patient must be awake, and not drowsy. The test must be stopped before the individual even approaches drowsiness.

Another problem is that of age. Fig. 6 shows the EEG of a 92-year-old woman who, we think, is the oldest normal person to have been recorded. Her EEG at 91 years of age, shown in

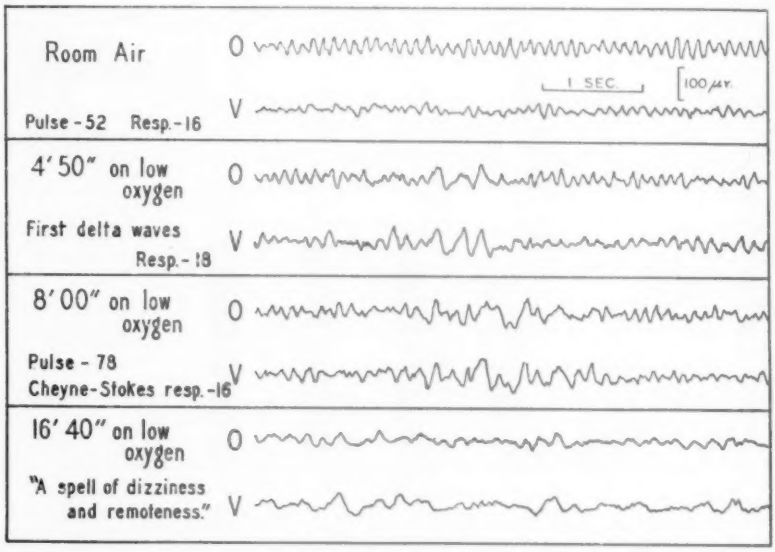

FIG. 4.—Simultaneous EEG's from the occiput (O) and the vertex (V) of a normal 41-year-old man breathing a gas mixture containing 8.8 per cent oxygen. The first slow ("delta") waves appeared at about the time that the subject became definitely cyanotic and also reported his first slight mental changes (7).

the two upper lines, cannot be distinguished from those of 25-year-old normals in our series. Age in itself, therefore, does not change the pattern. This woman had, of course, general arteriosclerosis, yet her pattern was still normal. A year later, when she began to show mental changes, such as slight retardation, occasional failure of recent memory, and confusion over names of members of her family, it was evident that her arteriosclerosis had begun to impair her cortical function. We took her EEG again at this time (third line in Fig. 6) and found that it had

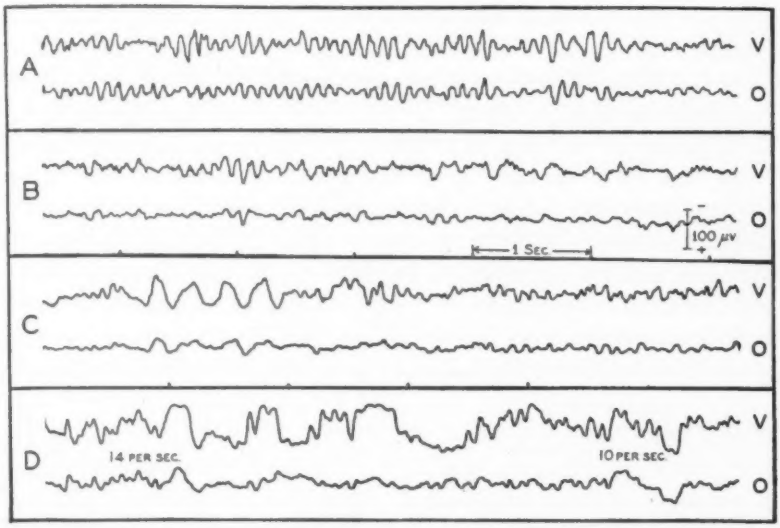

FIG. 5.—Simultaneous EEG's from the vertex (V) and the occiput (O) of a normal 41-year-old man during the onset of sleep. Same subject as Fig. 4. *A*, slightly drowsy; *B*, very drowsy; *C*, napping; *D*, asleep. [From (2) and (16).]

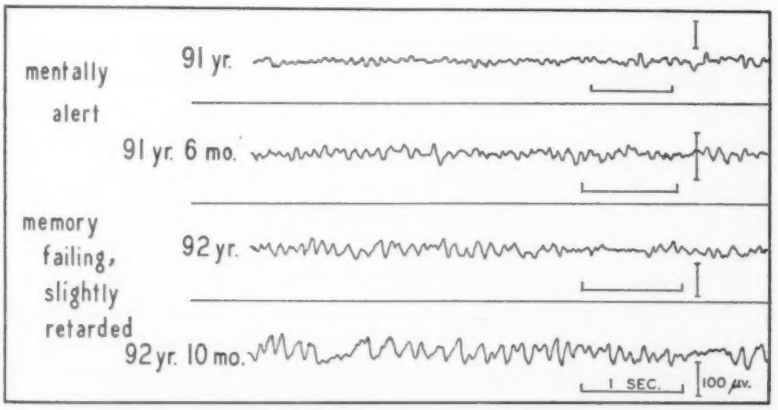

FIG. 6.—Occipital EEG's from an old lady on four different occasions. The subject has generalized arteriosclerosis but no psychosis. (Note the reduced sensitivity of the amplifier in the first record which causes an apparent difference between this and the second record.)

changed. Now, within two months of 93 years, the EEG has been recorded again, and the change is even more general, and in the same direction. The irregularity and slower waves are both more prominent in the last record. It must be emphasized, however, that this woman is in no way psychotic.

In Fig. 7 are records of four psychotic patients who are now senile, all of them younger than the previous case. The patient who is nearly 20 years younger, and whose mental condition is one of almost continuous confusion and whose diagnosis is "psychosis with cerebral arteriosclerosis," shows a record of greater irregularity and much slower waves. Evidently age in itself is not responsible for abnormal patterns. Fig. 1, illustrating the records of

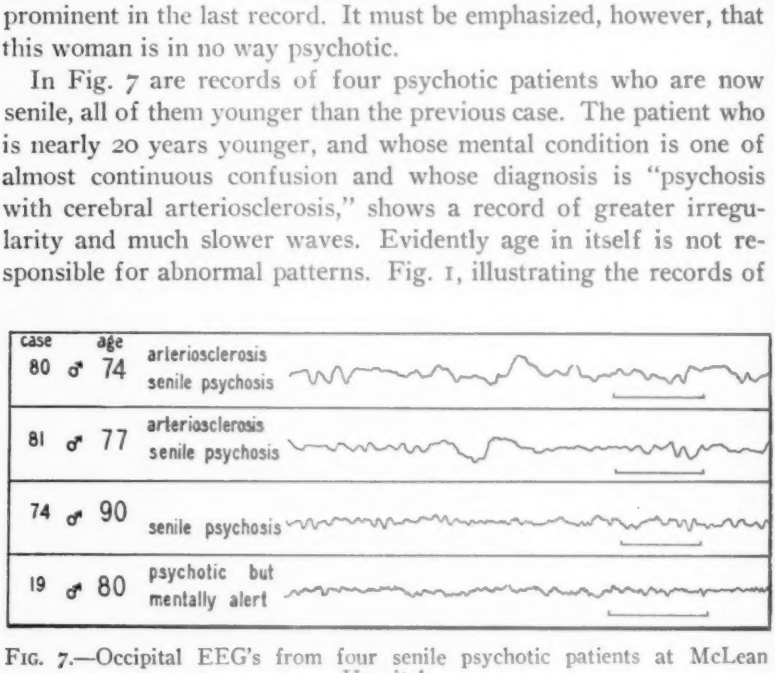

FIG. 7.—Occipital EEG's from four senile psychotic patients at McLean Hospital.

manic-depressive and schizophrenic patients, demonstrated that psychosis in itself does not produce such changes in the patterns. Arteriosclerosis, a pathological process, plus mental changes associated with it, is the common feature of the cases in Figs. 6 and 7.

Brain tumors modify the EEG in the region near the tumor (12, 6). The abnormal pattern is essentially permanent as long as the tumor remains. Unlike tumors, petit mal and grand mal epilepsy give characteristic abnormal patterns which come and go and may even change their foci or be found on all parts of the brain at the same time. These abnormal patterns may occur without clinical signs, but they are always present during seizures. Episodes of unusual behavior related to epilepsy, and there are many gradations, are associated with irregularities in the EEG which are sufficiently characteristic to be highly significant (13, 14, 15).

Aside from these specifically recognized abnormal patterns, we find records in which the electrical activity is totally different from the patterns found in our normal series. They show periods of greater irregularity of frequency and voltage. Slow waves appear spasmodically or intermittently throughout the record. "Spikes" suddenly occur singly or in groups. A burst of waves of one frequency quite different from the rest of the record may appear and continue even for minutes. The episodes stand out from the otherwise normal pattern. It may be said that when a record shows sections strikingly different from the main pattern, which either stop abruptly or peter out, merging into the normal pattern again, it is an indication that there has been a physiological disturbance in the brain.

The qualities of the episodes which break into the normal EEG have begun to take on meaning. Drs. Gibbs and Lennox have often correlated profound changes in the EEG, such as we have just described, with abnormal behavior of the patient at the moment of recording (13). An important relationship between such behavior and the EEG undoubtedly exists. This correlation serves as a background for evaluating the records of patients who may show no obvious abnormal behavior at the time of recording, but whose EEG's reveal the same types of changes.

The reason for discussing this side of the story in so much detail is that the records of psychotic patients which are different from the normal records, differ in just these respects. The difference is not in the fundamental inborn pattern which a person carries through life with him, but in the appearance of abnormal features which break into the pattern.

For example, Fig. 8 shows EEG's taken from the occipital and from the frontal regions of the head of a manic-depressive patient, once during her manic phase and twice while she was depressed. It will be observed that the occipital EEG does not change significantly. The first frontal record, however, differs profoundly from the other two in that it has large waves running at the rate of 6/sec. These waves emerged suddenly from the original pattern which resembled the two lower lines, continued for over a minute, and then returned to the original pattern. This was a clean-cut "episode" occurring in one area of the brain while another area, the occipital, was running true to form.

Fig. 9 compares samples from one record of a manic-depressive patient with samples from a record of a known epileptic. The epileptic has a type of petit mal pattern which is frequently seen unaccompanied by seizure (13). The large waves in the record of the manic-depressive occur at the same frequency as those of the epileptic, but do not have the characteristic spike. The middle lines in Fig. 9 compare quieter periods of cortical activity in the same two patients. The next to last line shows very slow waves in the

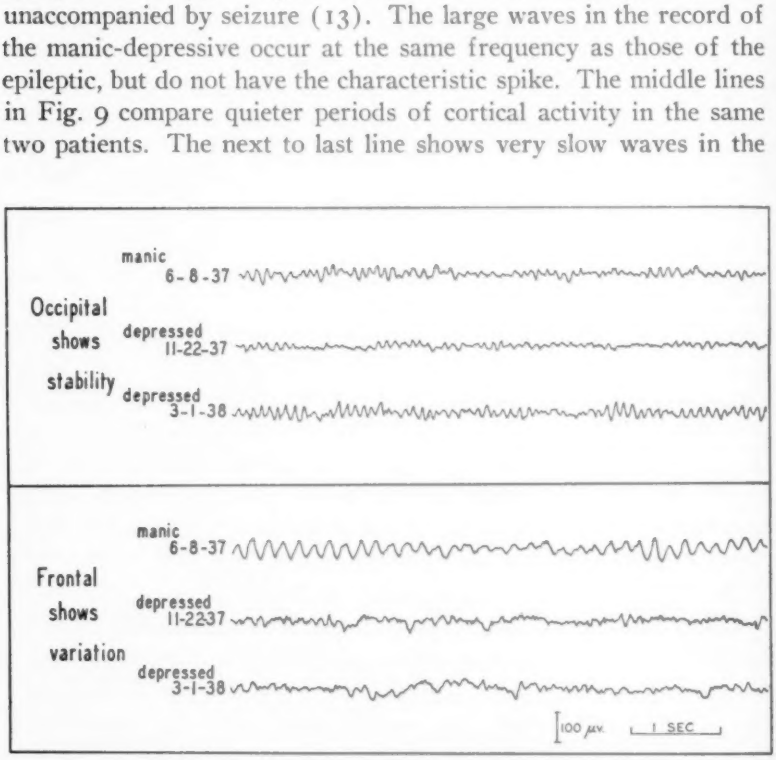

FIG. 8.—Occipital and frontal EEG's from a 33-year-old patient at McLean Hospital, taken on three different occasions. Clinical diagnosis: Manic-depressive psychosis.

record of the manic-depressive during a period of extreme confusion. Here his record is different from that of the epileptic, who was not confused at the time his record was being taken. We believe that whatever is disturbing the cortical function of this manic-depressive patient is of the same general nature and mechanism as that which disturbs the epileptic. The difference seems to be primarily one of degree.

Fig. 10 shows the record of a 50-year-old schizophrenic who has been a patient for 20 years at McLean Hospital. She has been

retarded since 18 or 20 years of age. She had sudden screaming spells two or three times a week for two or three years. Very recently she had two severe convulsions and was seriously ill, but recovered. We found a consistently localized area of abnormal activity low in the right temporal region.

These are only a few samples of what is being found within a mental-hospital group. We repeated the records of more than 100 patients at least twice, and many as often as seven and nine times, and one patient 23 times. The patients having atypical EEG's

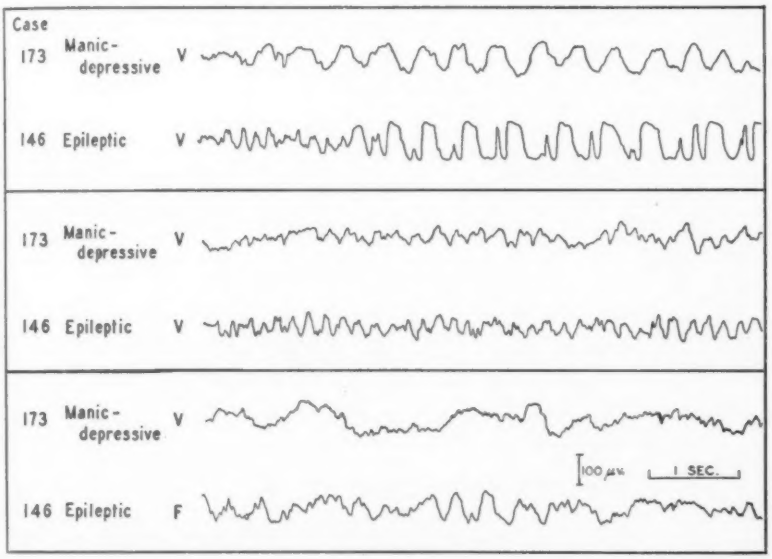

FIG. 9.—Comparison between the EEG of a 22-year-old manic-depressive patient during acute confusion, and the EEG of a 17-year-old patient known to have epilepsy, taken while he was clinically free of symptoms. V = EEG from vertex; F = EEG from frontal region. The three samples from the manic-depressive patient were nearly consecutive. Those from the epileptic were not continuous but are taken from a single record.

were found to include all of those diagnosed as epilepsy (there are seven of them) and all who were diagnosed as "psychosis with associated cerebral arteriosclerosis." The remaining atypical patterns are scattered indiscriminately among the schizophrenics, the manic-depressives, the psychoneurotics and other diagnostic groups.

For us it will be of great interest to know whether the removal of tumors or other lesions alters a patient's mental condition or

makes him more amenable to psychotherapy, psychoanalysis, or pharmacological treatments, and also whether those patients who do not respond to the various forms of treatment are the ones in whom we find atypical EEG's.

We have been following different groups of cases in coöperation with psychiatrists and analysts for two and three years. We believe that it would be of great advantage if they could know

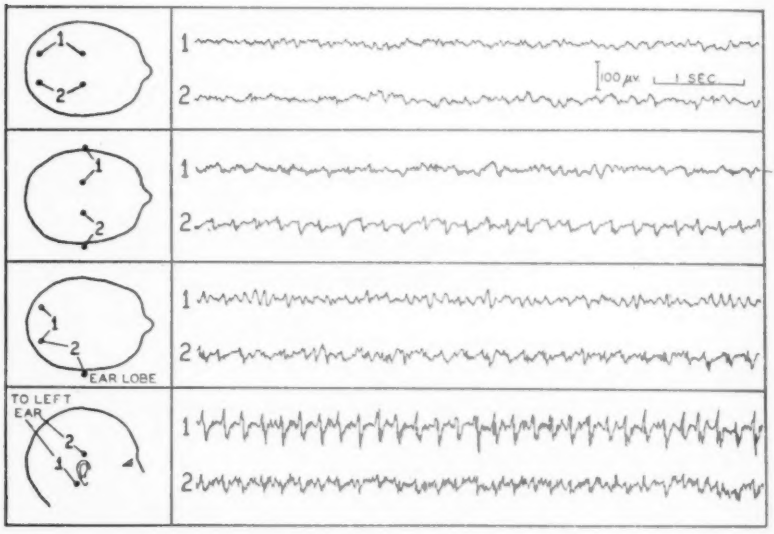

FIG. 10.—Four pairs of simultaneous EEG's from a 50-year-old patient at McLean Hospital, with diagnosis of chronic schizophrenia (see text). The placement of electrodes in each case is shown in the diagrams at the left. The abnormal activity appears consistently from the right side of the head, and most strongly when one electrode is placed on or just behind the right ear.

whether patients selected for intensive treatment, by psychotherapy, psychoanalysis, metrazol, narcosis, or insulin, show normal electrical activity of the brain, or whether there is evidence in the EEG of some physiological disturbance of the brain. Whether or not such changes in the EEG indicate lesions in the brain is an important field for further study. Moreover, it is of great interest to determine whether progressive changes in the EEG correspond with clinical and pathological evidence of progressive cerebral disease.

SUMMARY.

Electroencephalograms of 232 patients in mental hospitals, including 180 unselected cases at McLean Hospital and 52 chronic schizophrenics at Metropolitan State Hospital, have been studied since October, 1936. The fundamental patterns of these patients cannot be distinguished from those of our control series of 500 "normals." There is, however, a large proportion of patients whose records show variations outside of the normal range of variability (see Figs. 3 and 10). The abnormalities which break into the pattern, rather than the type of pattern itself, constitute the significant difference between the electroencephalograms of these patients and of normal individuals.

The quality of the atypical variations resembles that of the changes which occur in the electroencephalograms of persons known to have brain lesions, or who suffer from the various forms of epilepsy (Fig. 9), or of normal persons during sleep (Fig. 5), or when breathing an inadequate supply of oxygen (Fig. 4).

All the patients clinically diagnosed as having epilepsy or "psychosis with associated cerebral arteriosclerosis" showed abnormal variations, but it is significant that equally abnormal variations are distributed throughout all the other diagnostic groups.

BIBLIOGRAPHY.

1. Davis, H., and Davis, P. A.: Action potentials of the brain in normal persons and in normal states of cerebral activity. Arch. Neurol. and Psychiat., 36: 1214-1224, 1936.
2. Davis, H., Davis, P. A., Loomis, A. L., Harvey, E. N., and Hobart, G.: Human brain potentials during the onset of sleep. J. Neuro., 1: 24-38, 1938.
3. Davis, H.: Interpretation of the electrical activity of the brain. Am. J. Psychiat., 94: 825-834, 1938.
4. Hoagland, H., Cameron, D. E., and Rubin, M. A.: The electroencephalogram of schizophrenics during insulin treatments. The "delta index" as a clinical measure. Amer. J. Psychiat., 94: 183-208, 1937.
5. Jasper, H. H.: Electrical signs of cortical activity. Psychol. Bull., 34: 411-481, 1937.
6. Case, T. J., and Bucy, P. C.: The localization of cerebral cortical lesions by electroencephalography. Read at the meeting of the American Neurol. Association, May 1938.

7. Davis, P. A., Davis, H., and Thompson, J. W.: Progressive changes in the human electroencephalogram under low oxygen tension. Amer. J. Physiol. (Proc. Amer. Physiol. Soc.), 123: 51-52, 1938.
8. Loomis, A. L., Harvey, E. N., and Hobart, G. A.: Cerebral states during sleep, as studied by human brain potentials. J. Exper. Psychol., 21: 127-144, 1937.
9. Gibbs, F. A., Davis, H., and Lennox, W. G.: The electroencephalogram in epilepsy and in conditions of impaired consciousness. Arch. Neurol. and Psychiat., 34: 1133-1148, 1936.
10. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: The effect on the human electroencephalogram of certain drugs which influence nervous activity. Arch. Int. Med., 60: 154-166, 1937.
11. Berger, H.: Über das Elektrenkephalogramm des Menschen. VIII. Arch. f. Psychiat., 101: 452-469, 1933 (and other papers by the same title in the same journal).
12. Walter, W. G.: The electroencephalogram in cases of cerebral tumor. Proc. Roy. Soc. Med., 30: 579-598, 1937.
13. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Epilepsy: A paroxysmal cerebral dysrhythmia. Brain, 60: 377-388, 1937.
14. Golla, F., Graham, S., and Walter, W. G.: The electroencephalogram in epilepsy. J. Mental Sci., 83: 137-155, 1937.
15. Jasper, H. H., and Nichols, I. C.: Electrical signs of cortical function in epilepsy and allied disorders. Amer. J. Psychiat., 94: 835-850. 1938.
16. Davis, H.: The electroencephalogram. Tabulae Biologicae, 16: 116-131, 1938.

DISCUSSION.

DR. HERBERT H. JASPER (Providence, R. I.).—I have been very much pleased with this report of work in Boston, especially the clarity with which it was given. We have been working in Providence along similar lines, and rather independently on the same sort of patients. It is a pleasure to me, also, to see how closely our results correspond with those reported by Dr. and Mrs. Davis.

It should be obvious, it seems to me, that we have in the electroencephalogram an important tool for investigation of brain function, that the brain apparently is giving us a new symptom which can be used practically in diagnostic procedures. Certainly, however, there will be many years of hard work ahead of us before a thorough understanding of the significance of this new symptomatology is possible. In the presence of so many different types of abnormality one wonders whether the electroencephalographic records are of little importance when we find gross clinical changes not correlated with electroencephalographic changes, or whether in those psychiatric cases which formerly were considered as having no organic pathology, but which show pathology in the electroencephalogram, what is thus shown is really a significant factor in the disease.

The question of diagnosis has always bothered me as a foreigner in the field of psychiatry. It is very difficult for the physiologists to find anything

that will correlate with the clinical groups of the psychiatrist. We find signs of epileptic disturbances in schizophrenics; we find electroencephalographic changes in the paranoid or manic cases similar to what we find in some other organic brain diseases. This is all very confusing to the physiologist who attempts to work in psychiatry. Whether it is the fault of our physiological techniques or of the psychiatric methods of diagnosis, I will leave as an open question.

I would like to ask Mrs. Davis if she would be willing to say anything about the homogeneity of the psychiatric groupings in the light of electroencephalographic findings, and what does that imply with regard to such general shot-gun methods of treatment in psychiatry as the use of insulin or metrazol on large diagnostic groups.

DR. D. EWEN CAMERON (Worcester, Mass.).—I think the Davises have done a great service in indicating the extent to which it is now possible to correlate brain wave patterns with the psychoses.

My own feeling is that while it is now possible in a certain proportion of cases to demonstrate abnormalities in the brain wave pattern in psychotic cases, it is not yet possible, except perhaps in the case of epilepsy, to correlate this abnormality with the particular type of psychosis. Indeed, when one considers the way in which psychiatric classifications are made up, one thinks that in part it was based on prognostics, in part on some similarity between symptoms, and in a few cases on the basis of a definite knowledge of etiology.

The surprising thing would be that one should find an actual close correlation between pattern and diagnosis. I feel, too, that those who are working with the brain wave technique have to realize that when one is taking a record of a patient, let us say a schizophrenic patient, one is not taking a record of a simple, unified activity, but one is taking a record of a tremendous range of functions which are going on at the same time. The business of being a schizophrenic is not unitary, and consequently it is rather difficult to know what your pattern is correlating with.

I think that it might expedite the development of the brain wave work if one endeavored to set up situations in which one activity was predominant, for example, a situation in which the patient was in a state of tension, and contrast that with a state of relaxation, or condition of fatigue and contrast that with rest.

Animal experimentation might be used here to some advantage. It would be extremely interesting to know what goes on during the operation of a conditioned reflex, during a development of frustration, or in the course of the development of an experimental neurosis in an animal.

We have been fairly successful where we have kept at a simple level. Where we have given or taken away oxygen, or where we have withdrawn sugar progressively we have been able to make fairly definite correlations. But if we make a rather large jump and attempt to correlate to the top of the functional scale we are liable to get into difficulties.

We ourselves have been interested in trying to effect some correlations with emotional conditions, and have been able to detect a fairly definite and

predictable change in the record, the change being in the appearance of large waves when the person is stimulated emotionally.

My final point is that I agree with the Davises that it would be very interesting to know in our cases prior to treatment to what extent the brain wave record was damaged. I feel on the other hand that one must be on one's guard against assuming that if the record is damaged, even in its suggestion of atrophy, nothing more can be done for that patient. We know that in the case of tumors, with actual operative removal of brain issue, it is still possible to have a patient brought back to a very satisfactory level of performance.

Dr. Kenneth J. Tillotson (McLean Hospital, Waverly, Mass.).—As chief of the clinical service, I want to emphasize the great importance of the Davises' work reported in their paper. The paper is altogether too modest in a way, because from the clinical point of view the assistance that has been given to us is comparable to the assistance that the cardiologist gets from the electrocardiogram. It really is as definite as that in many cases.

I think the great danger, possibly, or the discouraging feature of work of this kind might be that one would try to draw too many general conclusions, and one might be confused by the questions that Dr. Jasper raises about diagnostic classifications. At McLean, we use the psychobiological approach and study the individual. We are prejudiced to any classificatory or categorical criteria. We try to study the patient as a psychosomatic individual or structure, and we feel that is the most fundamental approach, and one which has yielded a good deal.

The Davises have come to us and examined patients irrespective of any diagnosis, and usually examined them before they knew anything about the diagnosis. In some instances the graphs have shown very definite waves characteristic of an organic disturbance. In that way they have pointed out to us many conditions in which the symptoms of brain disease, from a neurological point of view, have been subclinical, and it has been found that there was a cyst or a tumor which has been confirmed by further electroencephalography and further clinical tests. This work has been done at the City Hospital and is a tremendous contribution in itself.

The type of work that Dr. and Mrs. Davis have done with us has been just the kind of work that every hospital staff needs, and I think it is definitely established that every hospital should have the electroencephalographic apparatus and careful workers like Dr. Davis, a physician and a physiologist, and like Mrs. Davis, who is not particularly interested in the classifications but who is willing to work on a single patient, taking as many as twenty-two observations over as long a period as necessary, night and day if need be, and who has done actually what Dr. Cameron has said he would like to see done on this clinical material.

She did not have time in her paper to emphasize the work she has done on emotional changes, the work done on the changing features of the psychotic patient or the non-psychotic patient. I think this work is probably the most

scientific and at the same time the most valuable to the clinician that we have had reported for a long time.

DR. W. G. LENNOX (Boston, Mass.).—I want to add my commendation to the discussion of this paper and to say also that the results which Dr. and Mrs. Davis have reported are similar to the results which Dr. and Mrs. Gibbs and I have obtained in a somewhat similar group of material.

In our work dealing primarily with the epileptic patients, we have encountered two rather disconcerting features for any close analysis of results. One is that you may have what looks like a bad record—that is, a lot of abnormal waves—in a patient who is essentially normal mentally. On the other hand, as Mrs. Davis has shown, there is a considerable proportion of these definitely abnormal mental patients who have normal records. Perhaps she could give some explanation why, if these waves are so significant, she may have normal waves in clinically abnormal patients.

With our epileptic group we now find that almost every patient does show abnormal waves even during periods when there are no epileptic attacks. But we have made it a routine to try to stimulate abnormality in such patients by having them perform a short time in a period of over-ventilation. This does bring out the abnormal waves in epileptics, and I wonder if such procedure has been followed with the schizophrenic patients with whom they have worked.

Not being a psychiatrist, clinical terminology is not sacred to me. It seems to me that words indicating various symptomatology are valuable as means of communication, but they should not stand in the way of more fundamental understanding of disorders when we can have the use of such a technique as the electroencephalogram.

I think Dr. Tillotson's comparison with the electrocardiograph is very close. You may have a patient with heart disease who has the symptom of dyspnea; another patient with the same type of heart disease may have edema. In either case you can greatly increase your knowledge or understanding of the condition if you have a record of the electrical activity going on in the heart. But you must recognize that you may have patients who show abnormalities in the electrocardiogram who do not show clinical symptoms, so that we must reconcile ourselves to viewing things from more than one angle, both from the clinical-pathological state and from the state of symptomatology.

MRS. PAULINE A. DAVIS (Closing).—To confirm what Dr. Tillotson said, I took great care for the first year and a half not to know anything about a patient, the diagnosis, or whether or not the patient was supposed to be difficult to handle.

Dr. Jasper mentioned the significance of the abnormal waves, and asked whether there is homogeneity in psychiatric groups. In the last 6 months, many patients presenting a history of night terrors or convulsions at the teething period in infancy, etc., were found to be those whose records had episodes in the pattern similar to episodes found in patients with convulsive

and epileptic disorders. There *may* thus be homogeneity in groups of patients having such symptomatology.

There is no correlation which I have been able to observe between EEG's and diagnoses of schizophrenia and manic-depressive disorders or other psychiatric diagnoses either in the McLean Hospital group or the Metropolitan State Hospital group. However, the EEG's of many schizophrenic patients show a striking similarity to records taken between seizures from epileptics. Some of us have a definite impression that epilepsy and schizophrenia are not mutually exclusive if one is judging the physiological activity of the cortex as seen in the EEG. Being a physiologist, the psychiatric diagnosis was not my concern.

Now, as to the implication of the abnormal—we have done a fair amount of physiological work where we deliberately upset the brain waves by upsetting the physiology of the normal brain, such as breathing low oxygen, or nitrogen, or giving sodium nitrite and various other drugs, and have produced abnormal patterns under these known conditions. Many experiments of this sort have given us excellent leads in interpreting the fact that the abnormal patterns among the psychotic patients may have something to do with the disturbed physiology.

The findings and the similarities to induced physiological disturbances were significant enough for the McLean Hospital staff to ask me to get an EEG of the insulin patients of Dr. Sakel and Dr. Jessner, and the metrazol patients of Dr. Sulzbach before treatment was started. I was not asked to record these patients until a schizophrenic patient undergoing metrazol treatment had a left-sided convulsion. The EEG records on this patient successfully localized extensive pathology. Encephalograms and X-rays of the skull revealed a porencephalic cyst of 7×5 cm. at the site of the abnormal EEG pattern. Treatment was discontinued as a result of the findings.

Dr. Cameron discussed emotional states, I believe. We have tried to check the emotional state of the individual carefully. We have run under standard conditions, but during the last year we have had a great many acutely disturbed patients. Even if it takes an hour for patients to get at ease, I take that time for the first record so that he or she will not be afraid of the surroundings or afraid of what is going to be done. Patients are often agitated and are sure they are to be burned in hell or killed. Each time a patient of this type comes in the behavior and remarks present the same stereotyped picture. One may call this emotional disturbance, but whatever it is, it is apparently not due to the surroundings. The mental state is in relation to the patient himself. An unfamiliar situation like being brought to the laboratory seems only to bring the mental state to the surface with the resultant stereotyped behavior. The EEG does not change when the patient eventually cooperates without this apparent emotional behavior. This may be quite aside from Dr. Cameron's point. A member of the staff attempted "emotional pin-pricking" on a case which was considered a good one for emotional reaction. We got most marked emotional responses clinically but the EEG did not change. One case, however, is not adequate for judgment.

Referring to Dr. Lennox's question as to why insane people can have normal records, and why abnormal records can be found among the normals, I may say that we may not know enough about the normals. For just this reason, we chose not to take average people but only people we knew over a period of about five years who we knew had no central nervous system disturbances. We even threw out people who had had infantile paralysis and had completely recovered. With experience we are carefully modifying our criteria.

There is an overlap, of course, but I think as we go on we will probably reduce the overlapping as we come to know the groups. Drs. Lennox and Gibbs and ourselves have what we call a "waste-basket group." We know the EEG's of some are not normal, but we don't know what they mean. It is one of the problems of the future to discover the significance of these atypical records.

CEREBRAL CORTICAL CALCIFICATION SIMULATING PICK'S DISEASE.

By EUGEN KAHN, M. D., ERNEST G. LION, M. D.,

AND

HARRY M. ZIMMERMAN, M. D.*

An opportunity recently presented itself to study thoroughly a patient who exhibited many intriguing symptoms. The findings in this case raised some problems of current interest.

CASE REPORT.

Chief Complaint.—C. T., hospital number 1506, male, 56, white, American born, Irish ancestry, Catholic, married, salesman, entered our clinic on January 8, 1937, with a complaint made by his wife that he was gradually becoming less and less efficient in his work.

Present Illness.—Starting in 1918, when the patient was 37 years of age, he became careless with the stock in his store. In 1926, at the age of 45, he began to develop hesitation and explosiveness in his speech. A year later, he started to show evidence of absent-mindedness. However, the most significant personality changes became most noticeable starting in 1933 after he failed in business. From the age of 52, in 1933, to the present (1937) there have been gradual but very definite personality changes.

His general intelligence was affected in that among other things he became absent-minded and forgetful. His attention and concentration became poor, while his insight diminished. His output of energy changed to overactivity by fits and starts. He became shy toward women. Temperamental changes were noticeable in the development of irritability, over-cheerfulness, coolness, facetiousness and inappropriate affect. Numerous changes in character took place. His attitude towards himself changed as he became reticent, shut-in, less conscientious, evasive, procrastinating, less proud, less neat, selfish and self-centered. His attitude towards reality changed with the development of day-dreaming, absent-mindedness, trance-like states and superstitiousness. He became less logical, less orderly, less methodical and less systematic. His attitude towards his environment became different, as he became shy, unsociable, stubborn, mistrustful, suspicious, grudge-bearing, seclusive, undependable, unkind, inconsiderate, infantile, peculiar, indifferent and dependent.

Since about 1933 hyperkinesis developed in the form of shrugging, fingering, tapping and cutting. Abnormal movements in the form of head nodding

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and neck spasms set in. Uncontrollable periods of laughter occurred. Restlessness developed in the patient. His movements became clumsy and it was readily observable in his writing, dressing and eating. There occurred a certain explosiveness in his body movements and speech.

Many changes in automatic functions have occurred in the later stages of the present illness. His night sleep became long but restless and fragmentary. Day drowsiness and napping set in. His appetite increased with the partaking of many meals a day. He developed the need of a rapid response to the urge for defecation. Increasing impotence has set in.

The patient's attitude toward his illness was characterized by indifference and lack of insight. The wife's attitude was characterized by great concern and solicitousness.

Family History.—The family history was essentially negative. His parents are dead. His sister, M. C. L., age 54, is the only sibling of the patient. She has no complaints, and a stereo-radiographic examination of her skull is negative.

Past Personal History.—The patient was born February 1, 1881, in Connecticut. He had a normal birth and early development. His progression through school was unremarkable, and he finished high school at the age of 18. He married in 1912; there have been no children. He was owner and salesman in a men's clothing store in which he did creditable work until the development of the present illness. His general health has been good. There has been no significant head injury. He had a thyroidectomy in 1923. In 1925 he developed cramps in his neck which have continued to the present at the rate of two or three each week, and have lasted for a few minutes at a time.

During his adult life, before the onset of the present illness, his personality was described as of average intelligence, above average energy output, even temperament, outgoing, ambitious and somewhat egocentric.

Mental Examination.—His appearance, general behavior and attitude were characterized by pleasantness, untidiness, unkemptness and indifference. He took care of his automatic needs. There was fragmentation of his long night sleep with day-drowsiness. His output of energy was increased by fits and starts. He was hyperactive and impatient. He showed a lack of initiative. Hesitation in responses was followed by explosiveness. His behavior showed a diminution of repressions and an egocentricity. His stream of speech and mental activity were characterized by a dearth of ideas with some perseveration. There was a lack of spontaneity. He was facetious. There was evasiveness with approximate answers. His chief preoccupation was with his speech disturbance. His emotional state was euphoric. *Witzelsucht* was present. His sensorium, mental grasp and capacity showed some decrements. His attention and concentration were impaired. His consciousness was clear. The patient's orientation was good. His recent and remote memory was impaired. The retention and recall was poor. In counting and calculation he was inferior. His general information and knowledge of current events were poor. He was defective in insight and judgment.

Physical Examination.—He was 5 feet 9 inches tall and weighed 177 pounds. His temperature, pulse and respirations were within average limits.

He was of a predominately pyknic physique. He had a few scattered skin lesions of the nature of keratosis senilis, angiomata senilis and fibroma molle. There were no significant angiomata on the face. He had a thyroidectomy scar. A complete upper denture and a partial lower denture were present. His peripheral blood vessels were soft, elastic and compressible. His blood pressure was 110/68. Examinations of the lungs, heart, abdomen and extremities were unremarkable. His left testis, epididymis and cord were lacking (probably an agenesis).

Examination of his cranial nerves revealed minor disturbances. Elsberg's olfactory tests showed marked diminution of sense of smell in both nostrils and a diminution of the trigeminal response to citral. The visual fields were normal. He had a myopia. His fundal vessels were normal. The patient's reflexes were equal and sluggish. There were no pathological reflexes. His sensation was undisturbed. He was clumsy in coordination. Abnormal movements were present in the form of transient tremor of tongue and coarse tremor of hands. Purposeless overactivity such as tapping with fingers, shuffling and fingering papers was present. Muscle spasms of the sternocleidomastoids developed occasionally when his neck suddenly turned. Electrical reactions of both sternocleidomastoids, however, were within normal range. No tetanic signs were present, nor developed with overbreathing. His speech was hesitating at the start and then became rapid, repetitive and explosive. Aphasic and apractic tests were all negative.

Laboratory findings.—Complete blood count, urinalysis, blood Kahn, blood bromide, stools and gastric analysis were not remarkable. The basal metabolic rate on January 29, 1937, was + 2 per cent, while on September 3, 1937, it was − 9 per cent.

Spinal fluid hydrodynamics, cellular content, Wassermann and colloidal gold reactions were not abnormal. Spinal fluid total protein, sugar, chlorides and cholesterol were within average limits. Spinal fluid calcium (4.28 mg. per cent was slightly below the lower limit of normal, while the spinal fluid phosphorous (2.11 mg. per cent) was within the upper limit of normal.

Bleeding time and coagulation were normal. Blood cholesterol, fatty acid, chlorides, total protein, non-protein nitrogen, serum total protein, serum albumin and serum globulin were within average limits. On three successive occasions in ten months' interval, the serum calcium was slightly low, in the vicinity of 7.22 mg. per cent, while the serum phosphorus was a little high, in the vicinity of 4.68 mg. per cent.

Radiographic Investigations (Dr. Hugh Wilson).—Stereo-radiographic examination of the skull on January 28, 1937, revealed an extensive amount of intracranial calcification overlying both cerebral hemispheres, and presenting a linear and convolutional pattern highly suggestive of cortical gyri. This calcification was fairly evenly distributed in frontal and temporal lobes with less extensive involvement in the parietal and occipital regions. The process was approximately symmetrical on both sides of the mid-line and had the appearance of calcification of the cerebral cortex. (Fig. 1.)

Encephalography on February 5, 1937, showed dilatation of the third and lateral ventricles, bilateral cerebral cortical calcification of the gyri, and

somewhat dilated sulci suggesting cortical atrophy were seen in the frontal lobes in close association with the cortical calcifications. (Fig. 2.)

A flat plate of abdomen on February 9, 1937, showed no evidence of intra-abdominal calcification. Radiographic examination of radius and ulna on February 9, 1937, showed no evidence of any bone changes.

Gastro-intestinal series on February 11, 1937, revealed second degree hypermotility of the small intestines.

Repeat roentgenographic examination of the skull on December 2, 1937, showed no change in the appearance of the skull with the exception of the operative defect, nor any change of the calcification in the cerebral cortex since the examination on January 28, 1937.

Clinical Psychological Examinations (Dr. Catharine C. Miles).—"Patient is a pleasant, cheerful, cooperative, apparently rather easy-going individual who displays marked defect in his speech and thinking. The pattern of mental capacity as revealed in the psychological studies is unusual in the range and character of the decrement. In general the defect has some resemblance to a case of marked frontal lobe deficiency that has been studied and also specific likenesses to other cases of definite central impairment."

Physiological Psychological Investigations (Dr. Walter R. Miles).—"The patient's verbal and motor responses are usually prompt but have the quality of automatism. He learns slowly or not at all on simple motor tasks. His immediate memory is defective, and on all types of tasks examined he shows grave defect in ability to perform mental integrations. This failure to synthesize impressions or meanings appears as a generalized central defect unrelated to any emotional element, and resembles the psychological behavior of the more incapacitated lobectomy cases uncomplicated by physical handicaps."

Brain Biopsy.—On March 19, 1937, under avertin and local anæsthesia, Dr. William J. German performed a biopsy on the patient. A button of bone about one inch in diameter was removed from the right frontal region. The subarachnoid space was found to be dilated in that region and the cortex appeared atrophic. From approximately area 8 of Brodmann a piece of cortex and subcortical tissue measuring about a half inch in diameter and one inch in depth was removed. Patient had a very satisfactory postoperative course.

Neuropathological Findings.—The gross specimen consisted of a block of cerebral cortical tissue measuring 2 cm. in length and 1 cm. in width. It did not appear to be unusual in any respect macroscopically. The leptomeninges were free from angiomatous change.

Microscopically, in Nissl preparations part of the cerebral cortex had an apparently perfect lamination. There were no ganglion cell changes of any description, and the cells were not decreased in number. The cortical and subcortical glia in these parts were normal in number and morphology. The lamination of other parts of the cortex, however, was completely destroyed by the presence of exceedingly large numbers of cylindrical bodies which were almost perfectly refractile by this staining method. (Fig. 3.) In hematoxylin-eosin preparations these bodies had a deep wine color and

FIG. 1.—Radiograph Showing Extensive Intracranial Calcification Especially Marked in Frontal and Temporal Lobes, and Presenting a Convolutional Pattern.

FIG. 2.—Encephalogram Shows Dilatation of Ventricles and Dilated Sulci in Frontal, Temporal and Somewhat Less in the Parietal Lobes Suggesting Cortical Atrophy.

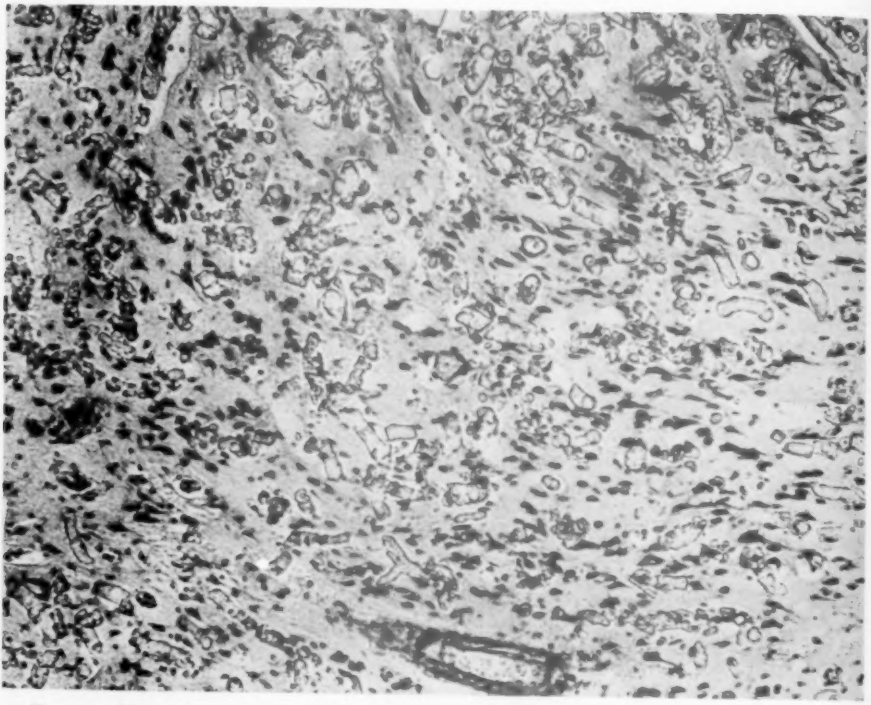

FIG. 3.—Photomicrograph of Frontal Lobe Cortex (Area 8 of Brodmann). Note Obliteration of Cortical Lamination by Refractile Calcific Bodies. Nissl Stain; × 125.

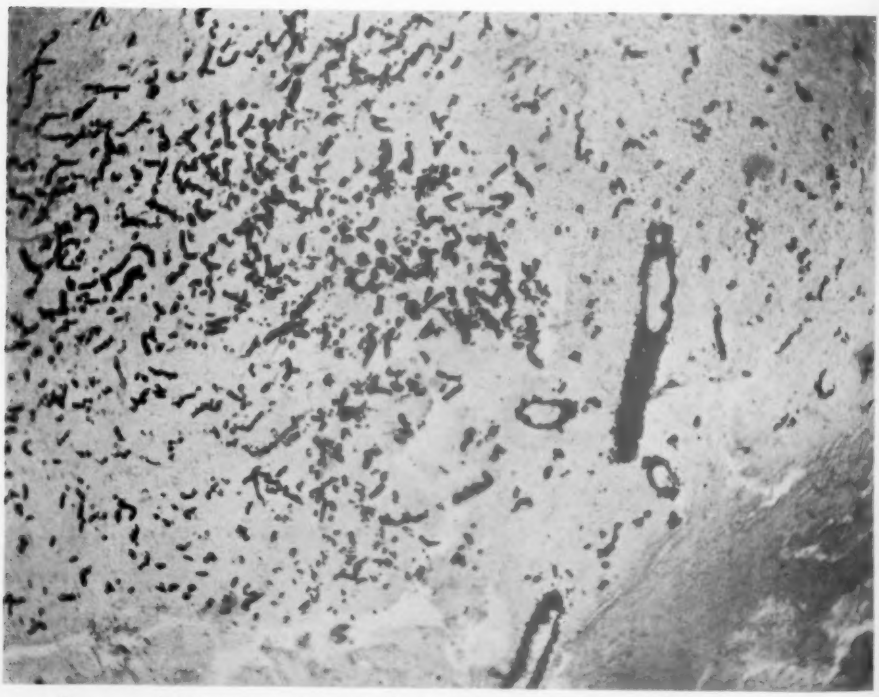

FIG. 4.—Calcification of Cortical Capillaries and Subcortical Arterioles. von Kossa Stain; × 45.

were readily identifiable as calcium deposits. In von Kossa preparations they were stained black. (Fig. 4.) After decalcification, the block of tissue still contained large amounts of iron pigment distributed along the capillaries.* This was proved by Turnbull's stain. The shape of these calcific deposits suggested that of the cortical capillaries, but a few vessels of this size were found without any calcification. The ganglion cells were reduced in number and those remaining had a wind-blown appearance which was the result of a loss of their polarity. There was a mild proliferation of the cortical glia and a more striking reaction of this variety was present in the subcortex.

Progress Notes.—His temperature, pulse and respirations have continued to be within average limits. His weight has varied from 175-180. The patient was always hungry and he ate many meals a day. He had daily bowel movements. Urinary and fecal urgency was present. He took care of his own organic needs. He bathed, dressed and shaved himself. There was carelessness about his clothing and his hair. He was ambulatory. Whenever he was disturbed his speech difficulty showed an exacerbation. He was very cooperative toward physicians and nurses. He took every opportunity possible to pun, and he showed some sound-associations. He did not question the purpose of procedures but he joked about them. Throughout his stay, he was passive, pleasant, untiring and submissive. He spent his spare time in the living room, but he did not speak or participate with the group of patients unless he was asked to do so.

Catamnestic Data.—The patient has been followed at frequent intervals over approximately a period of a year. Starting March 25, 1937, and for about the next eight months at about bimonthly intervals, the patient has had generalized epileptiform convulsions. Luminal therapy has not decreased their frequency. During this period there has been no essential change in his clinical picture. He has been able to take fair care of himself and to carry out simple tasks about the household. At the suggestion of the metabolic service, the patient was placed on calcium and viosterol therapy, but by the end of approximately a year, this had not altered the blood calcium level, nor the amount of cerebral calcification seen by radiography.

COMMENT.

This patient, born in 1881, manifested changes starting in 1918 which might have been the beginning of his present disturbance. However, it was not until 1933 following his failure in business, that definite personality changes became apparent.

These changes in personality included:

1. A decrease in social, cultural and ethical standards.

2. Sensorial-intellectual defects, namely, decrement of intelligence with marked deficiency in thinking, speech and reading

* The significance of this is discussed in a paper by Eaves.[2]

ability, impairment of recent memory and inability to perform mental integrations.

3. Loss of initiative, euphoria and *Witzelsucht*.

4. A tendency toward perseveration, hesitation coupled with explosiveness, and hyperactivity of an essentially purposeless sort (tapping, shuffling and cutting).

5. Morbid hunger and gastro-intestinal hypermotility.

The vast majority of the enumerated symptoms and signs have, according to recent investigations,[1] a rather direct relationship to the frontal lobes. We were, during the first period of our observation, inclined to diagnose Pick's disease in this patient. One of our psychological colleagues, Dr. C. C. Miles, emphasized the similarity of the findings in this patient with those in a case of marked frontal lobe atrophy which we had had the opportunity to observe. In this latter case there had been, in addition to the frontal lobe signs, temporal and brain stem manifestations; they had been considered in the final diagnosis of Pick's disease which was verified by autopsy. We think that a few of the signs in the present case are not strictly pathognomonic of frontal lobe disturbance.

The main point we want to make, however, is not one of special localization, but of the fact that there was, and incidentally still is, the clinical picture of a slowly progressing mental deterioration which closely simulates pictures observed in Pick's disease. The encephalograms showed a confluency of air in the frontal and temporal regions. But the cerebral calcification as ascertained by radiography and biopsy is fundamentally different from Pick's pathological process. Such an extensive deposition of calcium in the cerebral cortex is exceedingly rare. The calcification as far as can be seen covers essentially the areas in which Pick's disease is most often found, that is, the frontal and temporal lobes; it is less extensive in the parietal and occipital regions. Here our comparison ends.

Only vaguely can we speculate about the etiology of the case. We had been considering a possible causal relationship between the suspected removal of the parathyroid glands during the thyroidectomy and the calcification in the brain. We have been informed, however, by Dr. John P. Peters of the department of medicine that there is probably in this case a chronic latent tetany, but that there is no known condition in which reduction of the serum calcium is

associated with metastatic calcification. Dr. Peters would rather be inclined to consider the possibility that the calcification in the cortex in this case is secondary to a degenerative process. From the neuropathological point of view, one has to say that the small number of cells which so far have disappeared from the cortex do not warrant the diagnosis of a cortical atrophy.* One would rather surmise that the calcium deposition, wherever it came from and in whichever way it may have developed, was, and presumably still is, instrumental in choking a certain number of ganglion cells. If such an assumption were correct, the "atrophy" would be secondary to the calcification.

SUMMARY.

A case of mental deterioration has been described which showed essentially the picture of a frontal lobe disturbance simulating Pick's disease. Radiography revealed extensive symmetrical calcification in the cerebral cortex. Neuropathological examination of the brain tissue obtained by biopsy disclosed calcification associated with iron pigment deposition in the cortical capillaries, reduction in the number of ganglion cells and a mild proliferation of the glia.

BIBLIOGRAPHY.

1. (a) Kahn and Thompson: Concerning Pick's disease. Amer. J. Psychiat., 13: 937, 1934.
(b) Brickner, R. M.: The intellectual functions of the frontal lobes. Macmillan Co., N. Y., 1936.
(c) Fulton, Jacobsen, and Kennard: A note concerning the relation of frontal lobes to posture and forced grasping in monkeys. Brain, 55: 524, 1932.
(d) Levin, Paul: Restlessness and morbid hunger in man. Science, 84: 463, 1936.
(e) Levin, Max: Periodic somnolence and morbid hunger: A new syndrome. Brain, 59: 494, 1936.
(f) Mettler, F. A., Spindler, J., Mettler, C. C., and Combs, J. D.: Disturbances in gastro-intestinal function after localized ablations of cerebral cortex. Arch. Surg., 32: 618, 1936.
2. Eaves, E. C.: A contribution to the study of deposits containing calcium and iron in the brain. Brain, 49: 307, 1926.

* It is to be noted that the biopsy was obtained from a more posterior area in the frontal lobe than that which showed atrophy by encephalography.

EFFECTS OF VITAMIN B_1 IN SCHIZOPHRENIA.*

By L. H. CHASE, M. D., Worcester, Mass.

Several researches of recent years support the hypothesis that anomalous oxygen metabolism is a significant feature in schizophrenia.[1] The evidences include high incidence of depressed fasting oxygen consumption, lowered blood volume, retarded blood flow, increased blood lactic acid, and correlational discrepancies between oxygen and carbon dioxide of the blood. These findings have stimulated attempts to increase the rate of oxygen consumption of schizophrenics, and some tendency to clinical improvement has been reported after thyroid, dinitrophenol and dinitro-orthocresol medication.[2, 3]

There has long been general agreement that B_1-avitaminosis is accompanied by reduced oxygen utilization. More recently it has been shown that vitamin B_1 is an oxidative catalyst necessary for the normal intermediary metabolism of carbohydrates. It was considered possible, therefore, that administration of this vitamin in large doses might sufficiently augment the oxidation processes as to produce a beneficial clinical effect in schizophrenic patients.

A few previous studies on the effects of vitamin B_1 on nervous functions were encouraging. When inadequate diets in young children were improved with additions of vitamin B complex, Balken and Maurer [4] observed a gain in accomplishment in a series of psychological tests. Maurer and Tsai [5] showed that early deficiency of vitamin B complex was detrimental to the higher intellectual functions of experimental rats as demonstrated by maze-learning ability. Colby and her co-workers reported that increased amounts of vitamin B_1 accelerated infants' learning and maturation as indicated in basic behavior patterns and augmented alertness in attention and perception.[6]

Procedure.

Ten schizophrenic male patients on first admission were studied. Their ages varied from 17 to 30 years, with a mean of 20.1 years.

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The mean period of hospitalization at the beginning of the study was about three months. However, the duration of illness was estimated to have been from three months to seven years, with only one case of less than one year, and with durations of one to three years prevailing. Two patients were diagnosed as hebephrenic, four catatonic, two paranoid, one mixed, and one type undetermined. The periods of medication averaged eight weeks, with a range of six to nine weeks during which time each patient received 100 mg. of synthetic crystalline vitamin B_1 daily.* The medication was given orally except during the last two weeks, when intravenous administration was substituted. Immediately before and after the medication period a Stanford-Binet and various physiological tests were carried out.† These latter included the basal oxygen consumption rate and the levels of oxygen, carbon dioxide, lactic acid and glutathione in the venous blood. These were determined under basal conditions as were the pulse rates and blood-pressure which were recorded every other day throughout the study. Psychiatric observations were also recorded at least once weekly. Finally, three of the patients were given four intraspinal injections of the vitamin B_1 solution at weekly intervals, with the dosage gradually increased from 15 to 45 mg.

Results.

Of the 10 patients studied, nine showed either no significant change or some degree of progression of the schizophrenic process. One 17-year-old semi-stuporous catatonic subject improved progressively during the first month of treatment, was stationary during the second month, improved further after cessation of medication, leading to discharge from the hospital two months later. But since the amelioration had begun during the preliminary phase of the study before medication was instituted, it was regarded as merely coincidental.

The changes in the Stanford-Binet score appeared to be merely random, some patients showing increases and some decreases.

* We are indebted to Merck & Co. for liberal supplies of synthetic crystalline vitamin B_1 (Betabion).

† One patient was removed from the hospital just before the post-medicational test period.

Brief consideration of some of the somatic results of the experiment seems warranted, especially since the daily dosage of vitamin B was larger for the length of time given than has been hitherto reported.[7] No unfavorable effects, attributable to large dosage, were observed. The beneficial effect of vitamin B_1 on appetite is registered in the increase of the average weight of the ten patients from 62.7 to 64.2 kg. However, since similar improvement has been frequently reported with much smaller dosage (about 1 mg.), it would appear that very large doses have no advantage in this regard.

Only random fluctuation could be observed, individually and for the group, in oxygen consumption rate and in systolic and diastolic blood-pressure readings. With the exception of one patient who showed a significant rise of five beats, the pulse rate exhibited the same random fluctuation.

There was a slight and possibly significant decrease in the venous oxygen level of eight of nine patients in whom the tests were completed. The average changed from 11.6 to 10.5 vol. per cent. The venous carbon dioxide levels showed no significant changes. The oxygen-carbon dioxide relationship exhibited no abnormalities, either before or after treatment. All nine patients showed a statistically valid drop in blood lactic acid, the average being 1.4 mg. from the initial mean level of 12.8 mg. Blood glutathione levels showed merely random fluctuations.

In contrast to recent favorable reports of intrathecal vitamin B_1 therapy in other disturbances of the central nervous system,[8, 9] no significant clinical change was here noted in the three patients who received four weekly intraspinal injections.

In conclusion, it must be remembered that vitamin B_1 is a specific catalytic agent which influences the oxidation of carbohydrates at a single step in the oxidative sequence. Since this sequence involves an intricate series of chain reactions it is obvious that the study here reported by no means exhausts the possibilities of finding an effective therapeutic agent among oxidative catalysts.

Summary.

1. Ten schizophrenic male patients were given large doses of synthetic vitamin B_1 orally and intravenously for a period of eight weeks.

2. None showed clinical improvement that could be correlated with the medication. No deleterious effects were seen.

3. The Stanford-Binet scores showed only random changes.

4. No significant changes were observed in blood pressure, pulse rate, oxygen consumption rate, venous carbon dioxide and glutathione levels.

5. Decreases in venous oxygen and lactic acid levels were observed.

6. Three patients were given in addition four weekly injections of vitamin B_1 intrathecally without significant effects.

7. Extension of the study with other catalytic agents is regarded as desirable.

BIBLIOGRAPHY.

1. Hoskins, R. G.: Oxygen metabolism in schizophrenia. Arch. Neur. and Psych., 38: 1261, 1937.
2. Hoskins, R. G., and Sleeper, F. H.: The thyroid factor in dementia præcox. Am. J. Psychiat., 87: 411, 1930.
3. Looney, J. M., and Hoskins, R. G.: The therapeutic use of dinitrophenol and 3: 5 dinitro-ortho-cresol in schizophrenia. Am. Journ. Psychiat., 91: 1009, 1935.
4. Balken, E. R., and Maurer, S.: Variations of psychological measurements associated with increased vitamin B complex feeding in young children. Journ. Exper. Psychol., 17: 85, 1934.
5. Maurer, S., and Tsai, L. H.: The effect of partial depletion of vitamin B complex upon learning ability in rats. Journ. of Nutrition, 4: 507, 1931.
6. Colby, G. C., Macy, I. G., Poole, M. W., Hamil, B. M., and Cooley, T. B.: Relation of increased vitamin B (B_1) intake to mental and physical growth of infants: Preliminary report. Am. J. Dis. Children, 54: 699, 1937.
7. Peters, R. A.: The biochemical lesion in vitamin B deficiency. Lancet, 230[1]: 1161, 1936. Die Physiologie des Vitamins B_1. Dtsche. Med. Wchnschr., 63: 1144, 1937.
8. Friedemann, A.: Epidurale und endolumbale Anwendung des Vitamins B_1. Schweiz. Arch. f. Neur. u. Psych. Ergänzungsheft, 39: 236, 1937.
9. Stern, E. L.: The intraspinal (subarachnoid) injection of vitamin B_1 for the relief of intractable pain, and for inflammatory and degenerative diseases of the central nervous system. Preliminary report. Am. Journ. Surgery, 39: 495, 1938.

COURSE AND PROGNOSIS IN SCHIZOPHRENIA.*

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Considering the large quantity of publications on the various phases of schizophrenia, it is rather surprising to find so few well systematized studies and so little agreement in the conclusions reached on the course and prognosis of this disease. Recent reviews of the literature, particularly those by Langfeldt and Mayer-Gross, show this very conclusively, and in the present communication we should merely like to emphasize what appear to be the main reasons for these variations and the errors that have resulted in some of these studies. These are as follows:

1. *The Number of Cases Used.*—Some of the authors reported as few as 25-30 cases, obviously a number which is insufficient for statistical conclusions.

2. *The Period of Follow-Up Study.*—Patients who have been followed for only one or two years after discharge are not suitable for conclusions concerning the ultimate outcome of the disease.

3. *Lack of uniformity* in evaluating the original symptomatology as compared with the condition at the time of follow-up. The persistence or absence of original symptoms will depend upon the evaluation of this factor by the same observers.

4. *The Diagnosis.*—The criteria for diagnosis of schizophrenia may vary from one observer to another. A study of this type should include the criteria used by the author.

5. *The Adequacy of the Examination at the End of the Follow-Up Period.*—To be reliable this examination should be made by a psychiatrist, preferably one who has studied the cases originally. Reports by relatives, neighbors or non-psychiatric workers may introduce an important error.

6. *The type of treatment* used during the hospital stay. Patients who have received intensive treatment will naturally show a larger

* Read at the ninety-fourth annual meeting of The American Psychiatric Association, San Francisco, Calif., June 6-10, 1938.

proportion of good results than those who were simply observed in a hospital.

7. One should also take into consideration the effect of preconceived notions and the interests of the physician in a particular form of treatment.

It was with a view of avoiding the above-mentioned pitfalls in an investigation of this type, that the study was undertaken which we wish to report in this communication.

Procedure.

All the cases admitted to this hospital during the years 1929-1936 inclusive, in which the diagnosis of schizophrenia was made, were utilized for this study. All through this time one of the authors was in charge of the clinical work in the hospital and the cases used were personally examined by him and their treatment by the individual psychiatrists was carried out in cooperation with him. The records of these cases were analyzed and the important factors abstracted. In each case the subsequent course was ascertained and in April 1938 a personal re-examination of all the cases was undertaken by the authors assisted by the psychiatrists on the staff of the hospital. This was carried out at the place where the patient lived at that time, whether in a hospital or outside. Information was also obtained from family physicians, relatives and neighbors in regard to the present state of the patient and the adjustments made. No cases were used in which reports of the present condition were given by anyone but a physician.

Material.

During this period 344 patients admitted to the hospital were definitely diagnosed schizophrenic. Of these, 35 were not used because they could not be reached for psychiatric re-examination, although in some of them we had reports from other sources.* Three hundred nine patients were, therefore, actually used in this study. One hundred forty-seven of these were male and 162

* In most of these 35 patients the reports indicated that they were at least improved and some of them seemed to be recovered. This would mean that our results in terms of recovery and improvement were, if anything, too conservative.

female. All but four were natives of the state of Iowa. This was considered of importance in the evaluation of the results, since Iowa in common with the other states of the northern mid-west shows less of the racial, economic, cultural and other differences in its population than is the case in the industrial and coast areas. This ought to make a comparative study of the features of the disease and their influence upon the outcome less complicated and more reliable.

All of these patients have been studied and treated in the hospital for varying periods, the average being about 2½ months. The diagnoses were made shortly before the discharge of the patients at the staff conferences and the criteria used in making the diagnosis could be stated in the following definition:

Schizophrenia is a process disease, the causes of which are not adequately known but are most probably multiple in nature. It is characterized by:

A. Primary features consisting of (1) disturbances of *affect* such as inadequacy, inappropriateness, rigidity or dullness; (2) disturbances in *association* processes such as shortness, vagueness, lack of logical relationship, alliteration, neologisms, *vorbeireden,* etc.; (3) disturbances in *attention* such as shifts and gaps without apparent reason; (4) disturbances in *contact* with the outside world, withdrawal and autism.

B. Accessory features. (1) *delusions* either well-defined and therefore described under the particular category, or indefinite and described simply as present; (2) *hallucinations* described as above; (3) *motor* disturbances; (4) *mannerisms;* (5) impulsive behavior; (6) disturbances in *emotional* expression such as silliness, inappropriateness and ambivalence; (7) *confusion.*

Each case record was analyzed as to the presence and character of these features. In addition a number of other factors were included: age, sex, physical build, premorbid personality, intelligence quotient, somatic findings and permeability quotients. The historical data included heredity, civil status, previous attacks, onset, precipitating factors and the course in the hospital.

In the follow-up we noted whether subsequent attacks had occurred or whether the outcome was reached by uninterrupted progress. Finally, the results of the re-examination were tabulated in terms of improvement or lack of it. These factors were compiled separately for each case and then the material was separated first into two large divisions. (1) Group A consisted of all the cases who were discharged from the hospital before 1933, the follow-up of these covering approximately 5 to 9½ years. (2) Group B consisting of all 309 cases. In a third group were placed all the

patients admitted during the year of 1936 with a follow-up period of 16 to 28 months. These three general groups were then divided into sub-groups on the basis of their condition in April, 1938. Six such sub-groups resulted as follows:

I. *Complete Recoveries.*—The patient is living in the community practicing his previous occupation or one at about the same level. He is adjusting as he did before the onset of the disease and there is a complete lack of psychiatric symptomatology.

II. *Social Recoveries.*—The patient is living in the community, adjusting well socially, considered as normal by his associates, supporting himself independently, but there are some signs of minor defects mainly in his affect.

III. *Pronounced Improvement with Defect.*—The patient is living in the community, working and supporting himself, but at a lower level than previously and showing definite defect.

IV. *Slight Improvement with Defect.*—The process in these patients is arrested, but there is a pronounced defect which interferes with this adjustment and they need supervision.

V. *Unimproved.*—Most of these cases are in hospitals, although a surprisingly large number is out under supervision. They show no improvement or have gone on to deterioration.

VI. *Dead.*—Patients have died at any time since their admission to this hospital.

The various factors in the clinical picture, history and subsequent course were tabulated under these groups as follows:

Results.

In Table I, we present the relative proportions of the six subgroups as they are represented in the three major groups (A, B, 1936). In group A we find that the proportion of complete recoveries is only 14 per cent as compared with 17 per cent in group B and 31 per cent in the 1936 cases. It is of further interest to note the relative rates of complete recoveries in the cases with an *acute* onset of the disease. They are relatively higher in all the three major groups, but whereas in group A we have only 27 per cent, the 1936 group has 61 per cent of complete recoveries. As an additional check of the figures in group A (with a follow-up period of five years or more), we computed the proportions of sub-

groups in the cases discharged in 1929 and 1930, and found them to be practically identical to group A.

The analysis of the various factors noted in the records is presented in Tables II, III and IV. The data for groups A and B are separated and afford a good comparison, but for the purpose of establishing criteria only group A is used. The figures in these three tables represent in terms of percentage the relative frequency of the given factors within each sub-group. This of course may not afford an adequate picture of the absolute relationships of these factors within the total number of cases, since this could be better achieved on the basis of actual numbers. For lack of space, however, these figures cannot be included.*

TABLE I.

STATUS ON RE-EXAMINATION.

								Acute onset.	
	Total.	I.	II.	III.	IV.	V.	VI.	Total.	Recov.
A	177	25	14	6	13	102	17	33	9
		14%	8%	3%	7%	58%	10%		27%
B	309	53	29	12	21	173	21	64	23
		17%	9%	4%	7%	56%	7%		36%
1936	39	12	2	2	3	20	0	13	8
		31%	5%	5%	8%	51%			61%

Table II contains points of general information and in it we wish to emphasize the following points. The recoveries in group A show a preponderance of males over females, which is reversed in the unimproved cases. A comparison with the parallel subgroups in B shows that the recoveries are more numerous in the females than the males. The most significant feature in relationship to age is the relatively high proportion of persons in the second and fourth decades amongst the complete recoveries with a similar preponderance of those in the third and fifth decades in the unimproved. This relationship is further strengthened by the

* In evaluating the factors in these tables, we have found it most useful to emphasize the comparison of sub-groups I and V. This was done, firstly, because they are the most contrasting in outcome and, secondly, because they are the largest groups. It is for this reason that the order of the groups in the tables has been disarranged.

TABLE II.

GENERAL INFORMATION.

	A.						B.					
Group.	I.	V.	IV.	III.	II.	VI.	I.	V.	IV.	III.	II.	VI.
Number of cases.	(25)	(102)	(13)	(6)	(14)	(17)	(53)	(173)	(21)	(12)	(29)	(21)
Per cent of total.	14	58	7	3	8	10	17	56	7	4	9	7
Sex:												
Male	60	45	85	50	50	41	41	44	76	67	48	52
Female	40	55	15	50	50	59	59	56	24	33	52	48
Age:												
10-19	17	10	8	17	43	6	11	10	5	17	21	5
20-29	39	49	47	50	14	47	41	49	38	58	41	47
30-39	39	27	30	17	29	23	43	29	47	17	31	29
40-49	4	13	8	17	14	18	4	11	5	8	7	14
50 and over...	0	1	8	0	0	6	2	1	5	0	0	5
Physique:												
Leptosome	35	38	20	0	25	31	33	33	22	36	37	30
Athletic	26	18	10	20	17	12	15	12	6	9	11	15
Dysplastic	4	7	0	0	8	7	4	9	6	9	11	5
Normal	35	27	50	80	33	38	48	37	44	46	30	40
Question	9	8	30	20	17	7	10	6	17	9	7	5
Pyknotic	0	10	20	0	17	12	0	9	22	0	11	10
Personal:												
Normal	28	33	31	17	29	71	34	29	24	8	27	67
Cyclothymic ..	8	3	8	0	0	0	4	4	9	0	0	0
Schizoid	28	38	38	33	43	12	28	45	47	67	41	19
Intelligence:												
Above 105	36	8	0	0	8	0	20	9	7	10	13	17
90-105	6	31	43	67	31	20	13	27	43	40	33	17
70-89	29	28	43	33	46	40	35	37	43	30	33	33
Below 70	29	33	14	0	15	40	32	27	7	20	21	33
Scatter 5 years.	41	53	57	0	54	50	43	56	36	20	46	42
Somatic pathology.	32	20	31	17	36	35	34	16	29	8	28	33
Permeability:												
Decreased	50	36	36	67	33	31	33	35	32	25	26	29
Increased	17	10	28	0	17	8	16	10	16	13	15	12
Normal	33	54	36	33	50	61	51	54	52	62	59	59

fact that the socially recovered show tendencies similar to those of the completely recovered.

With regard to physical build, we find athletics and normals to have a slight preponderance in the recovered cases, whereas dysplastics and pyknotics are proportionally higher in the unimproved. Surprisingly, not one of the completely recovered cases was of pyknotic build. In the descriptions of the premorbid personalities, we have isolated three major types: (1) *normal,* a personality described by outsiders as not differing from the average; (2) *cyclothymic,* characterized by lability of mood with exaggerated reactions to success and failure; (3) *schizoid,* which was described as the well-known withdrawn, asocial and seclusive type. A number of other minor variations were excluded because they are not considered as adequate for statistical comparison and because of this the percentage in this part of the table does not add up to 100. We find here a preponderance of schizoids amongst the unimproved, whereas the cyclothymics are proportionally more frequent amongst the recoveries.

The intelligence quotients are computed on the basis of the short scale of the Stanford revision. Of interest here is the fact that there is a relatively high proportion of quotients above 105 in the recovered patients. At the same time there seems to be a noticeable preponderance of "scatter" of five years or more in the other groups.

Under somatic pathology we have noted the occurrence of any acute or chronic physical disease in the patients while under observation at the hospital. The highest proportion of these is found in the groups of the recovered cases and in group VI where perhaps the physical disease that was responsible for death had started early in the process.

The permeability quotients show a tendency of decreased permeabilities amongst the recovered cases and the reverse is true of the unimproved.

In Table III we have presented some of the data in the histories of the patients. In relationship to heredity, we have designated as *clear* those cases where the family history shows no mental abnormalities. Next we have the cases with one or more schizophrenics in the family, then patients where only one case of any psychiatric condition occurred in the family and finally those where

there were two or more. Here we wish to note the high percentage of clear histories in the unimproved cases as contrasted with heredity tainting in those that have recovered.

TABLE III.

HISTORY.

	A.						B.					
Group.	I.	V.	IV.	III.	II.	VI.	I.	V.	IV.	III.	II.	VI.
Heredity:												
Clear	36	46	30	60	54	47	50	50	38	50	43	42
Schizophrenic	12	12	8	0	0	12	11	10	14	10	7	14
One case	24	23	23	0	31	29	26	25	24	20	32	29
Two up	40	31	47	40	15	23	24	25	38	30	25	29
Unknown	0	0	0	20	8	0	0	0	0	20	3	0
Civil status:												
Married	45	33	27	75	56	60	51	31	26	43	48	53
Single	50	59	73	25	44	33	42	63	68	57	52	42
Divorced	5	3	0	0	0	7	5	3	5	0	0	5
Widow	0	5	0	0	0	0	2	3	0	0	0	0
Present attitude:												
Similar	12	11	8	50	7	0	15	9	9	33	10	5
Others	16	8	23	17	29	12	11	6	19	8	24	9
Onset:												
Gradual	64	87	77	67	77	82	56	87	71	67	83	81
Acute	36	13	23	33	23	18	44	13	29	33	17	19
Ppt. factors:												
None	8	26	47	33	14	29	11	26	33	25	17	29
Physical	32	23	23	17	29	41	26	20	29	8	27	33
Sexual	56	34	31	50	57	29	49	37	33	50	48	33
Social	28	27	8	33	43	23	25	26	19	33	34	19
Econom.	16	8	15	0	7	0	15	7	19	8	3	5
Course:												
Improved	40	11	30	17	35	6	36	7	24	8	27	14
Worse	20	41	23	33	29	76	23	38	29	25	17	67
Unchanged	40	48	47	50	35	18	41	55	47	67	55	19
Subs. attacks	8	25	8	33	0	18	4	24	9	17	0	19

The civil status of the patients was tabulated as single, married, divorced and widowed. To avoid possible error in computation we have obtained the average age of marriage of men and women in this state and used only those cases whose age was at or above

these averages. With this correction we find that in the groups of the unimproved and slightly improved cases the single people definitely outnumber the married, whereas the other groups show an opposite trend.

With regard to the previous attacks, we found that there is a higher proportion of attacks not similar to the present schizophrenic one amongst the recovered cases than in those unimproved.

With regard to the *onset* of the disease, we find a rather small percentage of acute onsets in our material. This is perhaps explained by the fact that what in some cases on the surface seemed to be an acute onset quite frequently was shown upon further analysis not to be so. We do, however, find that the proportion of acute onsets as compared with gradual ones is much higher amongst recovered cases than in those unimproved.

The data on precipitating factors were divided into the following types: (1) *none,* where no precipitating factors were revealed; (2) *physical,* where somatic disease, toxic agents or severe trauma seemed to be of importance; (3) *sexual,* where such factors as incompatibility in marriage, overt sexual conflicts, etc., seemed to precipitate the attack; (4) *social* and *economic,* which are self-explanatory. The complete and socially recovered cases showed the lowest proportions of "none," whereas they showed the highest of sexual and physical precipitating factors.

The course in the hospital showed some significant features in that sub-groups I and II had the highest percentage of improvements while still in our hospital, whereas sub-group V showed a very low percentage of improvements. Attacks following discharge from hospital were not reported amongst the socially recovered at all, were very rare amongst the cases that recovered completely, but were fairly frequent in those characterized as unimproved.*

In Table IV we have the analysis of the psychiatric symptomatology. Firstly, the *affect* was of course found to be disturbed in the large majority of the cases, since it has been taken as one

* It will be noted that no mention was made of the method of treatment. The reason for this is the fact that we have followed in practically all cases a more or less uniform procedure which in brief may be described as deep exploration (sometimes with the use of drugs), education and attempts at social readjustment.

of the most characteristic features of the disease, but whereas we find inadequate and inappropriate affect in the large majority

TABLE IV.

PSYCHIATRIC SYMPTOMATOLOGY.

	A.						B.					
Group.	I.	V.	IV.	III.	II.	VI.	I.	V.	IV.	III.	II.	VI.
Affect:												
Inad., Inap.	92	71	69	100	50	65	94	76	67	92	70	67
Rigid	4	1	0	0	0	12	4	1	0	0	0	14
Dull	4	24	23	0	50	12	2	19	24	8	24	10
Assoc.												
Disturbed	48	50	38	67	43	47	58	59	57	67	70	48
Mute	8	7	0	0	14	0	6	5	0	0	7	0
Attention	24	18	15	0	7	24	21	18	29	0	21	24
Delusions:												
Persecution	60	59	38	33	43	53	58	59	52	33	48	52
Grandeur	8	4	0	0	0	6	6	3	0	8	0	5
Jealousy	0	1	0	0	0	0	4	0.5	0	0	0	0
Religious	4	3	8	17	7	6	4	4	5	25	4	10
Present	0	11	31	0	21	0	8	14	19	0	10	5
Absent	28	24	23	50	29	35	23	21	24	33	24	29
Hallucinations:												
Present	8	7	15	17	14	6	11	8	10	8	14	5
Auditory	40	46	54	50	29	53	36	43	52	58	28	48
Somatic	4	4	8	0	0	6	6	3	10	0	0	5
Olfactory	8	4	0	0	0	0	6	3	5	0	0	0
Visual	4	12	15	17	0	12	4	12	14	8	7	10
Absent	28	34	31	33	50	35	38	32	33	33	45	43
Motor:												
Inactive	8	6	15	17	0	6	6	6	10	8	0	5
Resistive	16	11	0	0	14	6	8	8	0	8	7	10
Overactive	16	3	31	17	7	12	10	4	19	8	4	10
Contact	36	47	62	33	56	41	36	40	52	42	48	38
Mannerisms	20	17	23	0	21	11	17	17	19	17	14	19
Emotional	20	24	15	33	14	24	21	28	19	50	14	19
Impulsive	20	25	38	17	43	24	24	29	33	8	38	24
Confused	12	5	15	17	0	0	10	3	10	8	4	0

of the recovered cases the unimproved patients show a large proportion of the apathetic or dull affect. It is interesting to note that even a higher percentage of this type of disturbance was found

within the group socially recovered. The disturbances of *association* and *attention* do not seem to show any significant differences in distribution. This also seems to be true of *delusions* and *hallucinations,* with perhaps only one point of interest, namely, that the indefinite type of delusions grouped under the term *present* are only found in groups II, IV and V. Another factor of interest is that hallucinations of the visual type seem to be rare amongst recovered cases and fairly well represented in the others.

Motor disturbances were found in less than half of the cases and seemed to be more frequent amongst the complete recoveries. Poor or absent contact with a pronounced degree of introversion and autism was found more likely to occur in the unrecovered groups, whereas states of confusion seemed to have a higher representation amongst the recovered cases. The other secondary manifestations do not seem to show any significant differences in representation.

Comments.

A number of important features emerge as we analyze these results. First is the importance of a long enough period of follow-up study which is quite evident in our Table I. Here we see that the cases followed for five years showed only 14 per cent complete recoveries, as compared with 17 per cent in all of the cases. The significance of this rather small difference is enhanced by the fact that the 1936 cases yield 31 per cent complete recoveries. This means of course that with further observation this proportion will probably drop until it will reach the minimum obtained in group A. That this is actually the minimum is shown by the fact that the cases admitted in 1929 and 1930 show practically the same results as in group A. Added to this is the fact that cases with acute onset, when taken separately, show a remarkably high percentage of recoveries, particularly if they are followed for a short period of time. This finding is of particular importance when we come to evaluate the recent reports of treatment by insulin and metrazol. Frequently these reports have been made on the basis of follow-up studies which were even shorter than that in our 1936 group. Moreover, a great many of the reports on this form of treatment were made on the basis of selected material, most of the cases being of acute onset and short duration.

The relationships of the various factors tabulated in Tables II, III and IV and the ultimate outcome of the disease process show a number of significant features, but at the same time also throw some doubt on the dependability of some of these factors in the evaluation of the prognosis. Here we must again emphasize the fact that valuable conclusions can only be reached on the basis of the cases grouped under A, since in some of the cases that have been followed for a brief period of time and were found to be recovered there is no way of telling whether later on they will remain in the same group or shift to one of the others.

The distribution of the various groups amongst males and females and the difference that was brought out in the results may mean that following an early good improvement in female patients there is a subsequent tendency to shift toward the less markedly improved groups, whereas the males either improve less quickly or their improvement is more stable or both. The high proportion of patients of the third decade in sub-group V is not impressive enough to be of absolute significance, nevertheless this is in keeping with reports by other authors. Of further interest in relationship to age is the fact that schizophrenias developing after the fourth decade rarely show complete recovery, and, as is to be expected, they show a high incidence of deaths.

The high proportion of athletic and normal physical builds in sub-group I as contrasted with sub-group V is again in keeping with some reports in the literature. The surprising finding in this respect is the total absence of pyknotics in sub-group I which is in contradistinction to some reports in the literature. However, we must admit that all these differences are not high enough for reliable conclusions. This is also true of the pre-morbid personality types. We will agree with Langfeldt, however, in his conclusion that cyclothymic and extroverted personalities are more likely to influence the prognosis favorably than is pyknotic physical build alone.

The high proportion of intelligence quotients above 105 and somewhat lower proportion of pronounced scatter in sub-group I as compared with V may mean that in the less serious forms of the psychosis the deterioration, or what is more nearly correct, the interference with the ability to perform the tests, is less likely to occur and the performance is less spotty. Nevertheless, it would

mean, especially considering a previous report on deterioration (Malamud and Palmer) where the scatter was shown to be due to interference with directedness of thought processes, that it is a point to be taken into consideration in prognosis.

The higher percentage of somatic pathology and serological deviations in sub-group I would lead us to believe that cases which are going to recover are quite frequently associated with organic pathology, and where this is favorably influenced by treatment, deterioration is less likely to occur.

The factors in the life history of the patient seem to be of definite importance although not always in a positive fashion. The traditional belief that heredity is a factor of importance in schizophrenia may still hold as far as the psychosis as such is concerned but we certainly must agree with Langfeldt that it does not have a positive value in prognosis. If anything it seems to indicate that a positive family history is conducive to recovery rather than deterioration. This is seen from both the high percentage of clear family histories of the unimproved and incompletely recovered patients and, on the other hand, a rather high percentage of more than two mentally diseased members in the antecedents of the recovered cases. Of greater importance in this respect is the civil status of the patients. Here we find that the unimproved patients in sub-group V showed the greatest incidence of single people as contrasted with the high proportions of married people in sub-groups I, II, and III.

Another factor of importance is the manner of onset which I think should be logically discussed in connection with the precipitating factors. Briefly stated we may say that acute onsets show a higher percentage within the recovered and improved groups, whereas gradual onset is found proportionally more frequently among the unimproved. In keeping with this we find that definite precipitating factors, especially those of a physical nature and sexual maladjustment are found in greater proportions in sub-groups I and II than in the others whereas the absence of clear-cut precipitating factors are found in the highest proportion in sub-groups III, IV, and V. When we consider the fact that most of the available methods of treatment in schizophrenia depend upon analysis of life problems and attempts at social readjustment outside, these findings gain a particularly important significance.

The course at the hospital and the indications as to whether or not the process is yielding to treatment seem to be of definite significance in the prognosis, for the cases that showed complete recovery and, to a lesser extent, the socially recovered showed the highest proportion of improvement while still in hospital, whereas most of the unrecovered cases either did not improve at all or became worse. Finally the fact that very few or no subsequent attacks have been found in the recoveries would tend to strengthen the belief that these patients have shown a progressive course towards overcoming their disease.

It is somewhat discouraging to find that the psychiatric symptomatology as tabulated in our results affords very few if any points of importance in prognosis. The only factors that might be considered as significant are the types of disturbances of affect and contact and the motor symptoms. Apathetic affect and absence of contact with the outside is more frequent among unrecovered cases than among complete recoveries. To offset this, we find a high percentage of dull affect in the socially recovered cases. When we consider the fact, however, that whatever defects were found in the socially recovered patients were in the form of a poor affect and a tendency to seclusiveness our findings become not so surprising as they might seem.

Finally the high percentage of resistiveness, over-activity and confusion in the completely recovered cases would be in keeping with the generally accepted view that a stormy course and acute onset are favorable prognostic criteria.

Before we conclude we should like to draw attention to a few points that came to light in our study especially in relation to the general validity of the findings. In our personal interviews with the patients as was mentioned above, we were particularly struck with the lack of correlation between statements by relatives or friends and the actual status of the condition in some of the patients. This is further emphasized by a comparison of our figures and those of the present whereabouts of these patients. Of the 309 cases only 155 were either in mental hospitals or county homes, but we found 173 patients to be unimproved or worse. When we consider the fact that some of the patients in the state hospitals were really quite improved and working, some of them in responsible positions and remaining in the hospitals only because

they did not have anyone to whom they could be discharged, it becomes evident that a large number of the patients out in the community were not only not improved but were even worse. This was actually found to be the case and it throws a great deal of doubt on the validity of studies undertaken on the basis of considering patients out in the community as necessarily improved.

Summary and Conclusions.

1. This investigation was undertaken on all the patients definitely diagnosed schizophrenic that were admitted and treated in this hospital for a period of eight years (1929-36). The main purpose was to ascertain the ultimate course and outcome of the disease especially in relationship to data yielded by an analysis of the history, clinical picture and treatment.

2. The evaluation of the present status of the patients was made on the basis of personal re-examination by staff psychiatrists and the conditions were classified as: completely recovered, socially recovered, markedly improved, slightly improved, unimproved and dead. In order to assure adequacy and reliability of our conclusions as to ultimate outcome we have separated out the cases that have been followed up for five years or more (group *A*, 177 patients) from the total (group *B*, 309 cases) and our final conclusions are based primarily on a study of the former group. Comparison of these two groups and especially of group *A* and the patients admitted in 1936 showed that a follow-up period of five years is essential to obtain reliable results.

3. Our results were as follows:

(a) In group A the distribution of status at re-examinations was as follows: complete recoveries 14 per cent; social recoveries 8 per cent; marked improvements 3 per cent; slight improvements 7 per cent; unimproved 58 per cent; dead 10 per cent.

(b) Factors showing a relatively higher proportion within the group of completely recovered cases than in the group of the unimproved and, therefore, likely to influence the prognosis favorably are as follows: male sex, onset in second and fourth decades, normal and athletic physical types, cyclothymic personality, I. Q. above 105, somatic pathology, high I. Q., positive heredity, married civil status, previous attacks of a type different from the present,

acute onset, definite precipitating factors especially physical, sexual and economic, improvement while at our hospital, positive affect, motor overactivity and confusion.

(c) Factors showing a relatively higher proportion within the group of the unimproved cases than in the group of the completely recovered and, therefore, likely to influence the prognosis unfavorably are as follows: female sex, onset in the third decade and, particularly, after the age of 40, dysplastic and pyknotic physical build, schizoid personality, I. Q. below 100 and "scatter" over five years and more, single civil status, absence of precipitating factors, gradual onset, progressive course while at this hospital, dull apathetic affect, visual hallucinations, poor contact and autism.

(d) All the above factors (in b and c) taken individually are only more or less indicative, and not specifically characteristic of the ultimate outcome. Further intensive work on groups of definitely recovered and unimproved cases should be undertaken particularly for the purpose of ascertaining whether the occurrence of several of these factors in any given case is more significant as to prognosis, in other words, whether prognostic *patterns* can be established.

BIBLIOGRAPHY.

Bellinger, C. M.: Prognosis in schizophrenia, catatonic form. Psychiatric Quarterly, 6: 475-487, July, 1932.

Bleuler, E.: Primäre und secundäre symptome in der schizophrenie. Arch. f. Psychiatr., 90: 841, July, 1930.

Fromenty, L.: Les Rémissions dans la schizophrénie. Statistique sur leur frequence et leur durée avant l'insulinothérapie. L'Encéphale, XXXII: 275-286, Mai, 1937.

Fuller, R. G.: What happens to mental patients after discharge from hospital. Psychiatric Quarterly, 9: 95-104, January, 1935.

Henderson, D. K.: Discussion on the prognosis of psychoses occurring during adolescence. Brit. Med. Jour., 2: 1090-1095, Dec. 8, 1923.

Hunt, R. C., and Appel, K. E.: Prognosis in the psychoses lying midway between schizophrenia and manic-depressive psychosis. Am. Jour. Psychiat., 93: 313-339, September, 1936.

Langfeldt, G.: The prognosis in schizophrenia and the factors influencing the course of the disease. Humphrey Milford, London, 1937.

Lewis, A. J.: Prognosis in schizophrenia. Lancet, I: 339-341, February, 1935.

Malamud, W., and Palmer, E. M.: Intellectual deterioration in the psychoses. Arch. Neuro. and Psychiat., 39: 68. January, 1938.

May, J. V.: The dementia precox-schizophrenia problem. Am. Jour. Psychiat., IX: 401, November, 1931.

Mayer-Gross, W.: Prognose und Verlauf, in Handbuch der Geisteskrankheiten. Bd. IX. Die Schizophrenie. J. Springer, Berlin, 1932.

Strecker, E. A., and Willey, G. F.: Prognosis in schizophrenia. Jour. Ment. Sci., 73: 9-39, January, 1927.

Strecker, E. A.: Prognosis in schizophrenia. Assoc. for Research in Nerv. and Ment. Dis. Volume 10. Schizophrenia, pp. 119-189. Williams and Wilkins Co., Baltimore, 1931.

Sullivan, H. S.: The relation of onset to outcome in schizophrenia. Assoc. for Research in Nerv. and Ment. Dis., Vol. 10, Schizophrenia, pp. 111-118. Williams and Wilkins Co., Baltimore, 1931.

DISCUSSION.

Dr. Arthur P. Noyes (Norristown, Pa.).—It is somewhat strange that in such a frequent and serious a personality disturbance as we have in schizophrenia, there have never been any criteria or standards in determining the prognosis, or, better, any method formulated for following up such cases in determining the course and prognosis. Dr. Malamud has outlined a very definite method and he has applied it with the usual fine care that characterizes all his work. Of especial importance is the fact that he has outlined a method which may readily be used in following up these cases.

Another important fact is that he has applied the same method and criteria both in his original study and in the follow-up; that has perhaps been one of the weak points in previous studies of this kind. We have tried heretofore to compare data that could not be compared. Here we have a method in which the data should be capable of comparison.

As he also pointed out, all too often there has not been a long enough period between the initial diagnosis and the follow-up. This was well illustrated by the fact that he had 14 per cent of the totally recovered cases in those that had been out of the hospital for five years or more, and 37 per cent of totally recovered in those that had been out a year and a half to two years, and two years and a half, and 61 per cent in those that had an acute onset. We ought to bear these figures in mind when we study the results that are being secured with insulin and metrazol. I was rather surprised that when he took his earliest cases, he still found 14 per cent hadn't changed. My own prediction is that in ten years after the initial study, he would find a still smaller percentage than 14.

One thing that interested me was the finding that there was a higher percentage of complete recoveries in those having an I. Q. of over 105.

My own impression is that in many patients with a high I. Q. we found rather poor results. We can all think of cases, some of them cases of physicians, with rather outstanding attainments in many directions, who have developed schizophrenia of a very resistive type.

It is striking that the symptoms should shed so little light on the prognosis. I notice that the prognosis was much less favorable in those who had suffered

from visual hallucinations rather than auditory, and I should like to ask Dr. Malamud's explanation of that.

I am not surprised that he found that the prognosis is worse in women than in men. Perhaps that is natural, not merely because of adjustment in sex problems, but because of the blow to self-esteem in not being sought, and not fulfilling a purpose in having children; and I should like to ask why the prognosis was so much worse in those over forty.

Dr. Alfred Solomon (Chicago, Ill.).—I should like to ask what treatment was given these patients; if it was a spontaneous recovery, or he considers it such, it is very important. I have my doubts as to whether that occurs. I think that everything done to schizophrenic patients in one sense or another is treatment, whether or not it is intended to be so by the doctor who administers it.

Dr. Jacob Kasanin (Chicago, Ill.).—I am very much impressed with this excellent paper of Dr. Malamud and Dr. Render, and with the extraordinary hard work which they have put into it.

What happens to a schizophrenic patient is of paramount interest; and every psychiatrist interested in research has tried to follow up his own cases or a larger series of cases in a hospital, to find out what happens to them. While information of this type is very useful and gives us relative ideas of the results in the mass groups, I do not think that it will give us very much information about the prognosis in the individual case.

Dr. Malamud speaks of some cases in which the precipitating factors were of some importance. In a recent study by Dr. Campbell, of cases at the Boston Psychopathic Hospital, he found the precipitating or influencing factors were usually of extremely trivial nature; and this brings us to the very important consideration: What are the factors in both causation and treatment of schizophrenia, and especially its prognosis in the individual case, and it seems to me the answer to this question, which I hope Dr. Malamud will be able to pursue in his investigations, lies not so much in what the patient gives us in his adult history—that he has been employed or unemployed, or had trouble with his wife—but important psycho-emotional matters in his early life. It is only from these cases, not such a large series of them, but those of an intimate nature, that we can know how the personality has been developing in early childhood, and in this way find a reply to the question which interests us so much and which has, fortunately, occupied the attention of Dr. Malamud.

Dr. William Malamud (closing).—Dr. Noyes, I am much obliged to you for the very nice sentiments you expressed. Most of the questions Dr. Noyes asked I cannot answer—in fact, all of them. Certain data emerged as findings in my study. What they mean I do not know; I might say, however, that I very definitely emphasized at the end of the presentation that this study is only preliminary, to get an idea as to what guides we ought to follow, and now the important thing will be to study individual cases, and

it is quite possible that when we study individual cases, we will find out why people with high I. Q.'s show better prognoses, why people with visual hallucinations show poor prognoses, and why more men recover than women. One suggestion offered is that if we follow the cases for the whole group, we find there were more women than men in the complete recoveries. Those followed for five years showed more men than women. I suspect that complete recovery in a man may be slower but it is more stable.

We found that the customary diagnostic subdivisions—catatonic, hebephrenic, paranoid—showed absolutely no correlation with the prognosis, and I rather suspect that these subdivisions of schizophrenia are not distinct enough for statistical purposes.

How did we follow up our cases? That nearly ruined the hospital so far as both manpower and finances were concerned. It was extremely difficult to find these cases, but we tried our best, and Iowa, being a rather simple state, in comparison with some of the more complicated Eastern states, and perhaps Far Western states, we were able to get in touch with the majority of the cases. We have a fairly systematic program whereby these patients are followed every three months. Letters are written to find out where they are, and we at least learn what is happening to the patients, from the relatives.

Dr. Solomon asked about treatment. I have outlined the treatment in a paper I presented to the Association in 1931, "Psychotherapy in Schizophrenia." That is the most important treatment we use in the hospital, deep exploration combined with an attempt at readjustment. As to spontaneous recoveries, there aren't any, I think. As Dr. Solomon said, a patient in a hospital, whether because of what the physicians do for him or in spite of it, gets something out of the hospital stay and that is what helps in the recovery.

TREATMENT OF THE TOTAL ORGANISM IN SCHIZOPHRENIC PATIENTS.*

By WILLIAM CORWIN, M. D., Waltham, Mass.,
AND
JOHN W. THOMPSON, M. B., Ch. B., Cambridge, Mass.

Introduction.

The traditional concept in psychiatry has been that mental disease is the resultant of a pathologic or psychopathologic locus resident in the central nervous system. Some of the psychoses have been definitely correlated with diseased structures of the brain. Others, particularly schizophrenia have failed to be correlated definitely with specific changes in the brain tissues, despite the findings of some neuropathologists.

It is not unreasonable to suppose that some forms of mental disease might be the end result of a disturbance in function of some anatomical system or systems other than the central nervous system. This is a commonly accepted point of view in respect to diseases in other medical specialties. We know, for example, that heart failure can occur as a result of lobar pneumonia, anemia or nephritis.

It is part of our medical training to regard the organism as a composite of separate organs or systems and their functions, and to treat diseases of these organs from this point of view. A similar attitude has existed generally in psychiatry. For some years, however, the dualistic notion of mind-body relationship has been giving way to a monistic conception of the individual as a unit of integrated activities which exists in interdependent relations with its environment as well as with its component parts. From this point of view any restitutive efforts must necessarily be directed not towards the one organ which seems to be affected but towards the entire socio-individualistic economy. In the present study we have been particularly interested in the application of these concepts to the group of the so-called schizophrenic psychoses.

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In recent years increasing attention has been given to the factor of deficient oxygenation of the cortical cells in the schizophrenic states. This theory of anoxemia, so called, is supported by the presence in many schizophrenics, of a poorly developed and small heart, associated with a correspondingly smaller vascular tree.[1] The frequency of low blood pressure,[2] cyanosis of the extremities, and perhaps other vasomotor disturbances are well-known in these conditions; these we had thought might be associated with deficient oxygenation of the central nervous system. Furthermore, it has long been known to physiologists that diminution in the oxygen tension of inspired air can produce mental symptoms. Individuals exposed for long periods to low oxygen have shown reactions similar in some instances to states seen in schizophrenia. Barcroft's own experiences [3] and those of others to which he refers might be cited as examples of the dissociation, disturbed affective reactions and impaired insight and judgment that can occur under such conditions. The possible correlation of deficient oxygenation of the central nervous system and psychotic states has interested us and has led to certain experimental investigations. These have been chiefly the estimation of the gaseous content of venous and arterial blood, drawn from the jugular vein and femoral artery respectively, the simultaneous record of the type of respiration, and in some cases the determination of the cardiac output. This procedure has been carried out on a group of patients carrying the hospital diagnosis of schizophrenia, and has been reported in part elsewhere.[4]

It has appeared to us that the physiology of the schizophrenic is relatively fixed or set, one not readily adaptable to the constant changes of the internal and external environment. This unresponsive state has its psychological concomitant in the personality of the schizophrenics, aptly described as "unadaptable," withdrawing instinctively from all contacts presenting too great a demand, and, pursuing a groove-like type of existence.

Although these inquiries have had to do with the circulatory system and our first impression was that it was the important factor, we have more recently felt that there are other elements of equal importance. From the total organismal point of view, a disturbance of one organ or system leads inevitably to changes in the entire body, directly affecting the efficiency and state of

normalcy of the individual. It cannot be said that the heart is more necessary to the function of the individual as a unit than are the lungs or the kidneys or the nervous system. In attempting to understand disease, the tendency, however, is to concentrate on one organ or system and to isolate and treat, if possible, the unit that seems abnormal and which is taken as the cause of the individual's impaired function. Furthermore, it is our strong feeling that one may be dealing in cases of schizophrenia with systems which individually operate within normal limits but whose integration is defective; in other words, there may be errors of integration and not of systemic mal-function.

To study the true function of a physiological system, it is frequently necessary to obtain a series of observations indicative of that particular system's response to a given demand; the urea clearance test and the glucose tolerance curve are examples of such a procedure. If any one point on a "pre-diabetic" glucose tolerance curve is compared with one of a normal curve, the difference between the two may not be evident, but if the shapes of the entire curves be compared the abnormality is rendered more obvious. Thus far the "physiological microtome" which makes bio-chemical and physiological cross sections of the individual without regard for the variation in these observations with time as a coordinate, has demonstrated no essential difference between the schizophrenic and the normal individual; comparisons of resting blood sugars would be an example of such a procedure. More discrepancy is seen between the normal and the schizophrenic when they are made to adapt over a period of time and their mode of adaptation is recorded.

Viewed in this light, the treatment of schizophrenic patients should incorporate measures which would exercise those systems which are called into play whenever the organism is placed under stress and thus enhance integrative activity.

Method.

The patients have been subjected to a number of simple physical stresses; each type of stress being applied separately. In the first place, a series of graded exercises was instituted and adapted to the particular ability of individual cases. Until muscular proficiency

and physical condition progressed sufficiently the exercises were relatively simple and performed for the most part in the prone or supine position. The exercise periods were an hour in length and given morning and afternoon; the morning period was preceded by a cold shower and this was repeated after the exercises had been carried out. Frequent rest periods were encouraged. In several of the patients, another type of stress was used, namely, their exposure to air mixtures containing a low percentage of oxygen. The rationale for this procedure, differing from the one we now hold, was suggested by Quastel.[5] Thirdly, a certain limited amount of psychotherapy was used and each patient was seen frequently and rapport established in so far as possible.

Results.

The patients treated were all males who had been previously diagnosed schizophrenia, and included the several sub-types. Because of the case material available at the time, the first group treated consisted of patients regarded as chronic cases whose illness had existed as a rule for several years. Some degree of selection was made. The patients chosen were those in whom senile changes were not present and who were to some degree cooperative. The other two groups were unselected and were comprised of patients hospitalized on the whole for less than a year.

The results obtained from the method of therapy can be best evaluated by considering them as three groups. The criteria for regarding a patient as improved were increased interest and contact, decrease in apathy and untidy habits, change in mental content and psychotic ideation, development of insight, and improvement in conduct and social adaptation.

Group I.—This group consisted in 13 patients with long-standing, unimproved illness. The duration of mental symptoms varied from 1 to 11 years, averaging 6.1 years. The age varied from 19 to 35 years, averaging 27.2 years. Treatment was carried on for periods varying from six to nine months, although one patient was under treatment 14 months. This was followed by a period of at least two months without treatment. Of the group, one has been released from the hospital on indefinite visit, and remains out after a period of five months. Of the other 12, one is considerably improved,

three are moderately improved, four are slightly improved and four remain unimproved, showing no apparent change in mental condition. A harmful effect was seen in no patient. In three of the cases showing slight improvement brief periods of overactivity and uncooperation followed the period of improvement. They have since become quiet and cooperative following cessation of the treatment. The patients have now been off treatment for two months, and improvement persists in those cases which improved under treatment. In summary, of the 13 cases, two or 15 per cent showed considerable improvement, seven cases or 54 per cent slight to moderate improvement, and four cases or 31 per cent no improvement.

Group II.—This group consisted in four cases whose illness was of relatively short duration, varying from 3 to 20 months, and averaging eight months. The age varied from 18 to 26 years. Of this group, one has been sent home on indefinite visit after two and a half months' treatment. This patient was diagnosed hebephrenic type and was considered to be deteriorating rapidly. Another was considerably improved and is being considered for visit. These two patients received low oxygen inhalation in addition to graded exercise. Of the remaining two, one showed definite improvement, while the other showed no improvement. Treatment of the latter three has, up to the present, extended over a period of three and a half months. Bearing in mind the small number of cases in this group, we have 75 per cent improved and 25 unimproved.

Group III.—This group consisted in nine patients transferred to this hospital for treatment. Their length of illness varied from 5 to 39 months, averaging 12 months. The age varied from 16 to 35 years, averaging 23.2 years. Treatment has been carried on for one month, up to present writing. Of this group six show definite improvement and three remain unaffected and unchanged mentally, yielding percentages of 67 per cent improved and 33 per cent unimproved.

In all three groups, almost without exception, the physical condition was considerably improved as would be expected. This was evidenced by increase in weight (Table I), improved muscular tone, improved circulation as shown by a change from pale, sallow complexion to healthy, ruddy state, and disappearance of cyanosis and cold moist skin. Where low blood pressure was present, an

increase was noted. In some, acne vulgaris was present and was ameliorated. There have been no permanent untoward effects noted, and no phenomena dangerous to the life of the individual have been seen. On the contrary, the physical condition is always benefited rather than injured.

TABLE I.

TABLE OF INDIVIDUAL WEIGHTS OF PATIENTS BEFORE AND AFTER RECEIVING TREATMENT.

Subject.	Before.	After.	Difference.
1	120 pounds	147 pounds	23 pounds
2	136	152	16
3	128	148	20
4	140	133	−7
5	128	138	10
6	134	147	13
7	128	138	10
8	128	148	20
9	136	138	2
10	176	170	−6
11	123	136	13
12	132	157	25
13	116	116	0
14	95	108	13
15	145	150	5
16	182	192	10
17	130	130	0
18	126	148	22
19	140	150	10
20	165	178	13
21	132	150	18
22	138	162	24
23	122	125	3
24	142	162	20
Average	135	147	12

A few general observations may be made. The paranoid reaction-type has appeared to respond the least to the treatment. Good effects have been seen equally among the hebephrenic and catatonic types. There appears to be more favorable effects in the illness of shorter duration. When improvement takes place there seems to be a rhythmic cycle of improvement, regression and increasing im-

provement until a stationary condition obtains. Considerable individual attention is necessary in withdrawn patients to obtain the cooperation that is essential to the adequate performance of the exercises. Although low oxygen mixtures have been given to only a few patients, it seemed to have a synergistic effect. It is interesting and perhaps significant to note that the patient is quite confused mentally for several minutes after returning to room air from an atmosphere containing a low partial pressure of oxygen. Further observations and studies are being made with this procedure. It is our feeling that treatment should be maintained for a period of at least six months before considering the patient unresponsive to this type of therapy.

Conclusion.

A method of treatment in the schizophrenic patient is presented, based upon the concepts of organismal physiology wherein the efficient functioning of the individual is regarded as a total effect of the harmonious and interdependent reactions of all the organs and systems. These concepts do not permit of artificial and confusing separations into mind and body, and other psycho-physical parallelisms. The treatment is without danger to the individual and is, in fact, physically advantageous. In a total of 26 patients of varying periods of illness treated to date, 18 or 69 per cent showed improvement varying from slight to marked, while 31 per cent remained unimproved. Of the 18 who improved three or 17 per cent became more disturbed temporarily, but demonstrated an improved condition at the end of treatment.

Additional Note, November 1, 1938.

The progress of the patients months later is as follows:

Group I.—There has been essentially little change in the condition of these patients since cessation of treatment. The one patient allowed out on indefinite visit, remains on visit and is adjusting satisfactorily. One of the moderately improved patients is worse mentally at the present writing. The others maintain the state of improvement or unimprovement noted above.

Group II.—Of the four patients in this group, the one at home remains on visit and is doing well. The second patient has been

home for three months and is in good mental condition. The third patient is improved and is being considered for visit. The fourth shows slight improvement.

Group III.—Of this group, one has been home on visit for two months and is in good mental condition. Two others are considerably improved and are being considered for visit. One, formerly considered unimproved, is now slightly improved. Three, formerly improved, are worse, two being assaultive, and occasionally disturbed. Two of the three considered unimproved as above remain unimproved and had to be removed from the class because of their behavior. A tenth patient was added to the group and is in good mental condition at present and is being considered for indefinite visit.*

BIBLIOGRAPHY.

1. Lewis: Nerv. and Ment. Dis. Monog. Series, No. 35, 1923.
2. Freeman, H., Hoskins, R. G., and Sleeper, F. H.: Blood pressure in schizophrenia. Arch. Neurol. & Psychiat. *27*: 333-351, Feb. 1932.
3. Barcroft: The respiratory functions of the blood. Part I, p. 157. Cambridge Press, 1925.
4. Thompson, John W., Corwin, Wm., and Aste-Salazar, J. H.: Nature. 140: 1062, Dec. 18, 1937.
5. Quastel, J. H.: Personal communication.

* It is encouraging to note the reports of Dr. Myerson and of Myerson and Tillotson delivered to the Boston Society of Psychiatry and Neurology and the Massachusetts Psychiatric Society. These workers have applied the principles of the treatment outlined above and their results agree favorably with those reported here.

THE EFFECTS OF INSULIN HYPOGLYCEMIA ON THE BLOOD PRESSURE RESPONSE TO OXYGEN DEFICIENCY IN MAN.*

By S. H. KRAINES, M. D., and E. GELLHORN, M. D., Ph. D.†

Gellhorn, Ingraham and Moldavsky [1] showed recently that the blood pressure response to air-nitrogen mixtures is increased during insulin hypoglycemia. The rise of blood pressure produced by the inhalation of a gas mixture containing 6 per cent oxygen for one to three minutes was very slight in the narcotized dog at a normal blood sugar level, but increased progressively with falling concentration of blood sugar. When the original blood sugar level was re-established the blood pressure reaction to 6 per cent oxygen was similar to that observed prior to the injection of insulin.

The reaction studied is of importance for two reasons: (1) It gives added proof for the essential similarity between the effects of oxygen deficiency and those produced by insulin hypoglycemia (*cf.* also Glickman and Gellhorn [2]). (2) In view of the theory developed by Gellhorn [3, 4] that successful therapy of schizophrenia is based on excitation of the sympathetic centers brought about directly or indirectly by various procedures, the knowledge of procedures leading to marked sympathetic excitation is very important.

For these reasons it seemed important to establish the validity of these observations for the unanesthetized human subject. Experiments were carried out on psychotic patients to test the effect of the inhalation of oxygen-nitrogen gas mixtures on the blood pressure at various levels of blood sugar.

Materials and Methods.

The experiments were carried out on schizophrenic patients before the injection of insulin, during the hypoglycemic state and after

* Aided by a grant from the John and Mary Markle Foundation.

† From the Psychiatric Institute and the Department of Physiology of the University of Illinois, Chicago.

administration of sucrose by stomach tube or glucose intravenously. The gas mixtures were prepared by means of calibrated flow meters in Douglas bags from which the patient inhaled the gases; 8.1 per cent and 8.5 per cent oxygen were used for periods of five minutes. Special precautions were taken to avoid any admission of air from the outside. A mouthpiece was sealed with adhesive tape to the mouth and a noseclip was used. Pulse and respiratory rate and blood pressure were determined at one half minute intervals.

Although about one dozen patients were used in these experiments the report is based on six patients only, who were subjected to a total of 29 experiments. It was found that the records are reliable only when the patients are quiet and cooperative, since struggling will increase the blood pressure considerably even at a normal level of blood sugar. The patients were of both sexes; the ages varied between 18 and 30 years. The results were uniform for the whole group.

Results.

A typical experiment is reproduced in Fig. 1. In this case a control experiment was not carried out before the injection of insulin, but the test obtained after the injection of sucrose (which raised the blood sugar to 93 mg. per cent) may be considered as a control. The figure indicates that the blood pressure response to 8.1 per cent oxygen is greatly increased at 48.5 and 41.5 mg. per cent blood sugar and that the effects are completely reversible, as the control experiment obtained after sucrose injection indicates. That insulin as such does not influence the blood pressure reaction to a gas mixture low in oxygen unless the blood sugar is significantly lowered is seen in Fig. 2, in which a series of blood pressure tests obtained from the patient were recorded after 25 units of insulin had been injected. It is obvious from the record that insulin does not alter the blood pressure response to 8 per cent oxygen as long as the blood sugar level remains relatively high.

In order to avoid too severe sympathetic response we used 8.5 per cent oxygen in the majority of our experiments. The results were similar to those described for 8.1 per cent oxygen. They may be illustrated by Fig. 3, which gives a complete survey of the

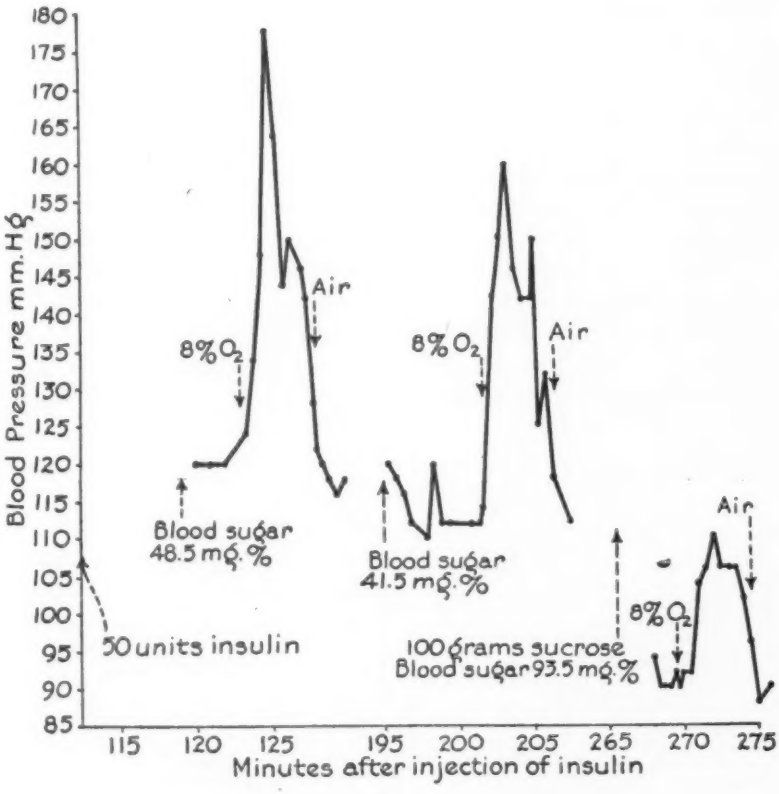
Blood Pressure mm. Hg.
180
175
170
165
160
155
150
145
140
135
130
125
120
115
110
105
100
95
90
85
8% O2
Air
8% O2
Air
Blood sugar 48.5 mg.%
Blood sugar 41.5 mg.%
50 units insulin
100 grams sucrose
Blood sugar 93.5 mg.%
8% O2
Air
115 120 125 195 200 205 265 270 275
Minutes after injection of insulin

FIG. 1.

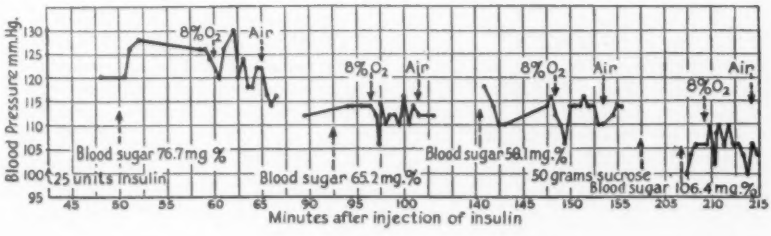
Blood Pressure mm.Hg.
130
125
120
115
110
105
100
95
8% O2
Air
8% O2
Air
8% O2
Air
8% O2
Air
Blood sugar 76.7 mg %
25 units insulin
Blood sugar 65.2 mg.%
Blood sugar 58.1 mg.%
50 grams sucrose
Blood sugar 106.4 mg.%
45 50 55 60 65 90 95 100 140 145 150 155 205 210 215
Minutes after injection of insulin

FIG. 2.

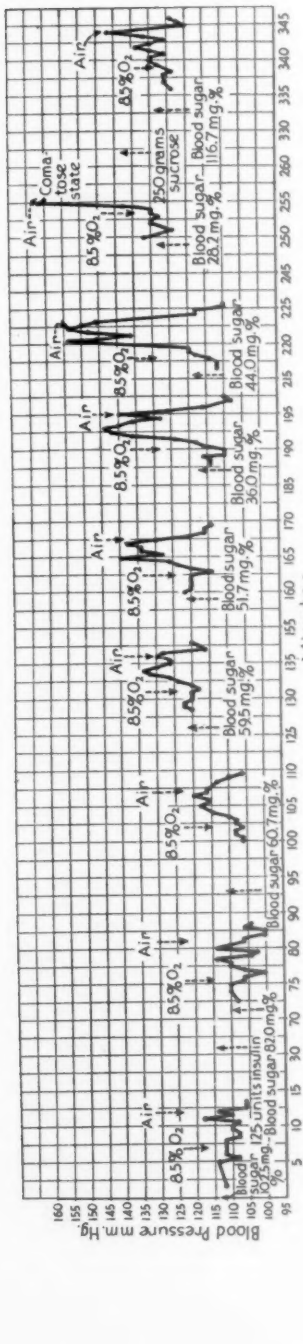
Blood Pressure m.m.Hg.
Minutes
Air
85%O2
Blood sugar 125 units insulin 0.25mg.%
Blood sugar 82.0mg.%
Blood sugar 60.7mg.%
Blood sugar 59.5 mg.%
Blood sugar 51.7mg.%
Blood sugar 36.0mg.%
Blood sugar 44.0mg.%
Blood sugar 28.2 mg.%
Comatose state
250 grams sucrose
Blood sugar 116.7mg.%

FIG. 3.

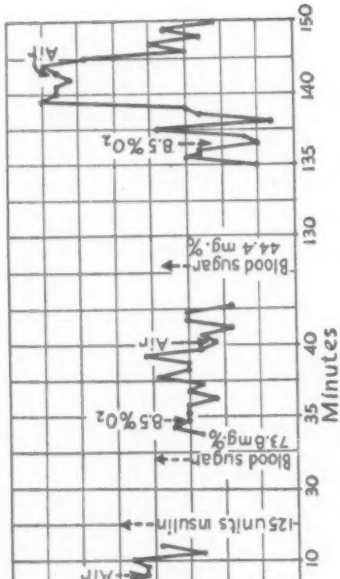
Blood Pressure m.m.Hg.
Minutes
Air
8.5%O2
125 units insulin
Blood sugar 97.2 mg.%
Blood sugar 73.8mg%
Blood sugar 44.4 mg.%

FIG. 4.

reaction during a four-hour period of hypoglycemia. It is evident from the curves that the blood pressure response to 8.5 per cent oxygen is practically the same before the administration of insulin (blood sugar 102.5 mg. per cent) and at a level of 82, 62 and 59.5 mg. per cent blood sugar which we observed during the first one and one-half hours following the injection of insulin. The next test, carried out when the blood sugar had fallen to 51.7 mg. per cent, shows a slight but distinct rise in the response of the blood pressure to 8.5 per cent oxygen. Much greater reactions were observed, however, when the blood sugar fell to 36 and 44 mg. per cent. In the last reaction recorded during hypoglycemia the blood sugar had fallen to 28 mg. per cent. The blood pressure rose sharply on inhalation of 8.5 per cent oxygen, but the experiment had to be discontinued after three minutes because of convulsions, thereby precluding a comparison with the earlier experiments. After this test, sugar was given by nasal catheter and the blood sugar level was restored to normal. The twitchings disappeared and the reflexes reappeared. The patient reacted again to call. A final test with 8.5 per cent oxygen was made one and one-quarter hours after the administration of sucrose. The result (Fig. 3) shows a complete return to the normal reaction.

Fig. 4 illustrates the dependence of the blood pressure response to anoxia on the blood sugar level by the records obtained from another patient. The blood pressure response to 8.5 per cent oxygen was the same before the administration of insulin and 30 minutes later, when the blood sugar was 73.8 mg. per cent. When, however, the blood sugar fell to 44.4 mg. per cent the reaction was greatly increased, as in the experiments previously described.

From these and other experiments it is evident that the increase in the blood pressure response to anoxia occurs whenever the blood sugar concentration falls below a critical level. This is in agreement with our experiences with dogs. The result is best illustrated by Fig. 5, which shows the blood pressure response to 8.5 per cent oxygen of the same patient in a series of experiments in which different amounts of insulin were administered. Obviously, the response is increased whenever the blood sugar falls below 48 mg. per cent. The results obtained with other patients were similar.

Another fact of some importance is recorded in Fig. 5. The records were obtained in 10 experiments carried out on the same

patient, in which single doses of from 50 to 1000 units of insulin were administered. Although the number of observations carried out with excessive insulin doses is small, Fig. 5 tends to show that the response to oxygen deficiency, as far as the blood pressure reaction is concerned, is more marked when insulin is injected in

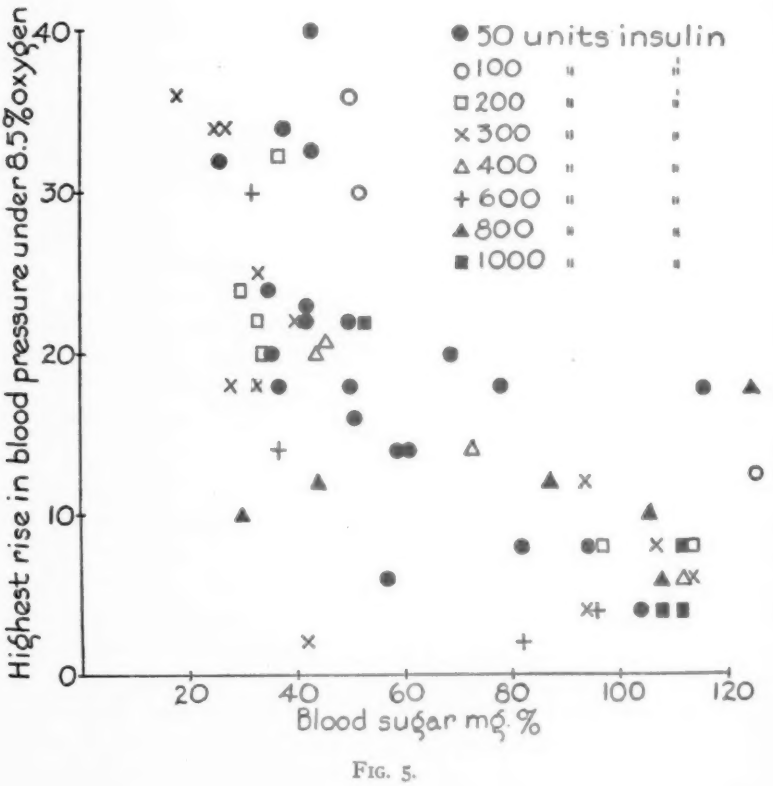

FIG. 5.

moderate amounts than when it is administered in excessive amounts. This statement refers, of course, to the reaction observed at similar blood sugar levels as Fig. 5 indicates.

In our previous investigation on the influence of hypoglycemia on the blood pressure reaction of dogs to gas mixtures low in oxygen tension, it was observed that the reaction depends not only on the blood sugar level but also on the duration of the hypoglycemic

condition. It was found that when a low blood sugar level was maintained for some time the sensitivity to oxygen deficiency as measured by the blood pressure response was greatly increased. This observation could be duplicated in our experiments on human subjects only in relatively few cases (Table I) and was not pronounced. In other cases the reaction remained almost unchanged.

During the course of insulin hypoglycemia the blood pressure may show some variations, but the reaction studied in this paper occurs irrespective of such (rather slight) variations.

As has already been mentioned, the reaction may be observed at low blood sugar levels produced by single insulin doses varying between 50 and 1000 units. The very large doses of insulin seem

TABLE I.

INFLUENCE OF THE DURATION OF THE HYPOGLYCEMIC STATE ON THE BLOOD PRESSURE RESPONSE TO 8.5 PER CENT OXYGEN.

Time (min. after injection of insulin).	Blood sugar mg. %.	Max. rise of blood pressure during anoxia in mm.	Remarks.
101	32.7	22	Before insulin
131	29.7	24	(200 units)
161	33.7	18	blood pressure
190	34.7	32	response was 8 mm.

to diminish the reaction somewhat. They had, moreover, a very striking additional effect, at least in one patient who had been subjected to a large range of insulin doses. As Table II indicates, insulin administered to patient B in doses of 50 units or less did not produce any convulsive movements or coma. With doses of 150 U and more such movements occurred regularly; they consisted initially of jerking of the small muscles of the hands and spread gradually to all other muscle groups. These phenomena were associated with clouding of consciousness, which later deepened into coma. Table II shows that the twitching movements occur earlier with increasing doses of insulin. This fact is clearly seen in Table II, although the values seem to be modified somewhat by the interval in which the injections had been given.

Despite the fact that such large single doses as 1000 units were given, no unusual effect was noticed. There were no subjective or

objective effects that differed from those with the use of smaller doses. The coma which tended to come on more quickly at 1000 units (one and three-quarter hours) than at 125 units (three and three-quarter hours) was relieved just as quickly by sucrose.

One patient in coma after 800 units had been administered returned to consciousness when 5 gm. of sucrose was injected slowly into the mouth, following which he was able to drink the rest of the sugar solution.

The patients were given sucrose as soon as coma appeared, for the jerky movements prevented the taking of accurate blood pressure readings.

TABLE II.

INSULIN DOSAGE AND ONSET OF CONVULSIONS ON PATIENT B.

Date.	Insulin dosage.	Onset of coma.
7/1/38.....	50 U.	No convulsions or coma.
7/5/38.....	125 U.	3 hrs. 40 min.
7/6/38.....	200 U.	3 hrs. 20 min.
7/7/38.....	300 U.	2 hrs. 7 min.
7/12/38.....	450 U.	2 hrs. 10 min.
7/13/38.....	600 U.	1 hr. 53 min.
7/15/38.....	800 U.	1 hr. 17 min.
7/19/39.....	1000 U.	1 hr. 32 min.
7/26/38.....	300 U.	1 hr. 42 min.

It was our practice at the beginning of the experiments to give the patient 2 gm. of sugar for each unit of insulin injected. However, the patients would vomit whenever 300 or more gms. were given at one time, following which they would become hypoglycemic and need intravenous administration of glucose. Later it was found that two glasses (400 cc.) of 50 per cent sucrose solution by tube was preferable, and a glass full was then given orally about every hour to the point of tolerance by the patient. The convulsions and coma occurred at rather widely different blood sugar levels, which did not show any direct relationship to the number of units of insulin administered.

Summary and Conclusions.

Experiments on schizophrenic patients were carried out on the influence of the inhalation of 8.1 and 8.5 per cent oxygen on the blood pressure at normal levels of blood sugar and during the hypoglycemic state induced by insulin. The experiments confirm the earlier work of Gellhorn, Ingraham and Moldavsky. It is found that the blood pressure rises only slightly on the inhalation of 8.5 per cent oxygen for five minutes when the blood sugar is normal, but that the rise increases with progressively falling blood sugar. The critical blood sugar level below which the blood sugar response is markedly increased is at about 50 mg. per cent. The reaction is completely reversible upon administration of sucrose and is independent of the blood pressure level. With excessive doses of insulin it is less pronounced than with moderate doses, up to 200 or 300 units. Excessive doses of insulin decrease the latent period of insulin convulsions.

BIBLIOGRAPHY.

1. Gellhorn, E., Ingraham, R., and Moldavsky, L.: The influence of hypoglycemia on the sensitivity of the central nervous system to oxygen want. Jr. Neurophysiol., 1: 301-312, 1938.
2. Glickman, N., and Gellhorn, E.: The effect of oxygen deficiency on the sensitivity of rats to insulin. Am. Jl. Physiol., 121: 358-363, 1938.
3. Gellhorn, E.: The action of hypoglycemia on the central nervous system and the problem of schizophrenia from the physiologic point of view. Jl. Am. Med. Assoc., 110: 1433-44, 1938.
4. Gellhorn, E.: Effects of hypoglycemia and anoxia on the central nervous system. A basis for a rational therapy of schizophrenia. Arch. Neurol. Psychiat., 40: 125-146, 1938.

A PUBLIC HEALTH APPROACH TO THE PROBLEM OF CONVULSIVE DISORDERS.

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AND

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It is the intention in the present paper to describe briefly the nature and extent of the problem of convulsive disorders as it exists in Ontario and to indicate the manner in which an attempt has been made to devise an effective public health approach to the problem.

The responsibilities of a modern Department of Health are many and varied. The amount of emphasis given to any one disorder or group of disorders must, of necessity, be determined by the nature of the illness and the frequency of its occurrence. That convulsive disorders are of a nature to warrant serious attention, no one will deny. The dramatic nature of the seizures themselves, the social and occupational handicap which results from the mere threat of their occurrence, the intellectual and emotional disintegration which so often follows their persistent recurrence —these are among the factors which make convulsive disorders a matter of major social and economic as well as medical significance.

While the significance of convulsive disorders in this sense is generally admitted, the frequency with which they are to be found in the population is not nearly so well recognized. The exact number of persons suffering from such disorders in any large community can only be determined by direct survey. However, a fairly accurate estimate may be made on the basis of the results of such surveys carried out on large populations elsewhere. Since proper perspective in a public health program calls for information on the relative incidence of various disorders, this question becomes a matter of considerable importance.

In 1931, Pollock [1] called attention to the wide discrepancy between the rate of .62 epileptics per 1000 of population reported by Rosanoff for Nassau County in New York State and the much higher rate of 5.15 epileptics per 1000 enlisted men reported by the Surgeon-General. Pollock concluded that the true incidence would probably be found somewhere between these two extremes. According to Thom,[2] Tracy found a rate of 4 epileptics per 1000 children in the Boston school population. More recently, a British report [3] issued by His Majesty's Stationery Office in 1936 gives the results of a survey covering an area with a population of half a million, on the basis of which the incidence of epilepsy is estimated at 1.8 per 1000. Anderson [4] in 1936, reported a rate of 2.1 per 1000 of general population for nine counties surveyed in the State of Michigan.

In the light of the available evidence from such field surveys, a conservative estimate of the rate of convulsive disorders in the general population may be set at about 2 per 1000. Applying this rate to Ontario which has a total population of just under three and three-quarter millions, we may conclude that there are approximately 7000 persons in the Province suffering from convulsive disorders.

The significance of such an estimated incidence is twofold. In the first place, it is noteworthy that there are as many persons in the Province suffering from convulsive disorders as there are persons suffering from active tuberculosis. Hence, it seems evident that convulsive disorders occur with sufficient frequency to warrant aggressive public health attention.

In the second place, it is of interest to note that there are in Ontario, less than 1000 epileptics in institutions. Thus, it is further evident that the problem presented is not exclusively, or even mainly, one of providing institutional treatment. If 85 per cent of the persons suffering from convulsive disorders are to be found in the community, then it is toward the community that our attention must be directed.

Progress in any aspect of public health has always been accompanied by a shift in emphasis from the remedial treatment of the products of disease as found in hospital, to the preventive treatment of illness in its earliest stages as found in the community.

There seems every reason to believe that the problem of convulsive disorders calls for a similar approach.

In 1930, the Province of Ontario organized a number of travelling Mental Health Clinics which, since that time, have operated as mobile out-patient departments from Provincial Mental Hospitals. These clinics are staffed and equipped to provide a psychiatric consultation service to physicians in the community. They have as their primary objective the early recognition and preventive treatment of persons showing indications of mental illness.

During their eight years of operation there have been referred to these clinics 937 cases in which the problem presented was that of convulsive seizures. A study of the records of this rather large group of cases has provided us with useful information concerning the nature of the problem as it exists in the community, and it may be of value to report briefly certain of the observations which have been made.

Sex.—Fifty-seven per cent of the cases were male. This finding is probably no more than a reflection of the somewhat greater readiness on the part of the community to refer males which has been observed also in connection with other problems.

Age.—The cases ranged all the way from infancy to old age. The distribution according to age is of interest, however, in that it shows a very abrupt rise in frequency during the first school years (ages 5 and 6) followed by a more gradual but continued increase up to a peak at ages 14 and 15. Thereafter, there is a steady decrease in the frequency of cases.

It is of interest, also, that 10 per cent of the whole group were under five years of age when referred to clinic. The fact that such a substantial group of pre-school children were referred indicates that the provision of a consultation service of this type in the community does actually assure access to cases at an early stage of illness.

A little over one-half of the group were of school age. Yet investigation showed that relatively few of these cases were receiving a suitable type of school training. Many of them were struggling unsuccessfully with classroom work beyond their ability. Nearly one-quarter of this group had been excluded from school because of their seizures and were receiving no formal academic training whatever.

Of the whole group of more than 900 cases, nearly 300 were adults. In many of these older cases, convulsive seizures had been a problem for years and the difficulties encountered in treatment were thereby much increased. However, the presence of this group indicates clearly that the problem of convulsive disorders as it exists in the community is in no sense limited to any special age group.

Intelligence.—Psychometric examination in these cases showed that 47 per cent were mentally defective, 29 per cent were borderline or dull normal and 24 per cent were normal or superior. Thus, as a group, these cases are handicapped not only by convulsive seizures but also by limited ability. Yet, the fact that less than half of the group were found to be mentally defective is in marked contrast with comparable observations on institutionalized epileptics. According to these, from 80 to 90 per cent of patients are usually reported to be defective in intelligence either because of native defect or, more often, as a consequence of frequent and repeated seizures over a long period of time.

It must be remembered that institutionalized epileptics constitute a highly selected group representing usually the end-products of a long period of inadequately controlled seizures. A survey of 1000 cases in one large hospital revealed that the average duration of seizures had been 13.6 years. If it is true that severe and repeated seizures result in both organic and functional deterioration, and if it is also true that, in most cases, seizures can be controlled by proper treatment in the early stages of illness, it is evident that such treatment should be instituted as early as possible. Furthermore, such treatment is preventive in a very significant sense if it serves to lessen the amount of deterioration and so reduce the handicap which such deterioration involves.

Referring Agent.—In three-quarters of the cases, the problem was referred to the clinic by the family physician, the school nurse or some other medical agency in the community. From this it can be inferred that the cases under consideration probably represent a stage of illness intermediate between the chronic institutionalized epileptic and the relatively mild case encountered in general practice which reacts favorably to routine treatment. For the most part, these are cases in which ordinary treatment measures have failed to take effect and further special assistance is required.

Associated Problems.—The reasons given for referring the cases are of interest. "Convulsive seizures" is given as the sole reason in less than half the cases. In the remainder, various associated problems are cited, the most frequent of which are "backwardness at school" and "suspected mental defect." Speech defect, enuresis, emotional instability and violent outbursts of temper are also mentioned in a large number of cases. It is interesting that, although psychotic manifestations are very seldom noted in these cases, milder personality changes, difficulties in emotional control and unsatisfactory social adjustment are observed in the majority of them. Again, the early control of seizures becomes essentially preventive if it makes possible the preservation of an integrated personality and the elimination of the unhealthy habits of emotional expression so frequently associated with this type of disorder.

Treatment.—After examination and investigation, the clinics found it necessary to recommend institutionalization in only 25 per cent of all cases of convulsive disorders referred to them. When it is remembered that, especially in the first few years of operation, many of the cases referred were already chronic epileptics of long standing, this proportion is strikingly low. The fact that, in three-quarters of the cases, it was found possible to institute therapeutic measures in the community which served to control the seizures, suggests the extent to which the community treatment of convulsive disorders has been found practicable.

While establishing control of the seizures, is, of course, the first objective in treatment, this has not usually proved to be the most difficult aspect of treatment. To establish control is one thing—to maintain it is another. This latter aspect has presented the greater difficulty since it involves sustained co-operation both of the patient and of the patient's family which is often not easy to secure. But having succeeded in establishing and maintaining control of the seizures, there then remain for treatment the complicated personality problems which have usually developed in association with them.

Any attempt to evaluate the degree of success achieved in the preventive community treatment of convulsive disorders in Ontario would be premature at the present time. However, the attempt to approach the problem in this way has already taught us much

concerning the difficulties to be faced. On the basis of the evidence so far obtained the following conclusions seem warranted:

(1) Preventive treatment in the community is not only desirable but is definitely feasible in a significant proportion of those persons suffering from convulsive disorders.

(2) Such treatment, to be effective, requires the direct and continued supervision of a trained field staff working in close co-operation with the family physician. That such an arrangement can be developed is shown by the fact that, during 1937, at the request of the physicians concerned, more than 100 of these cases were carried under the continued supervision and direct treatment of the clinics.

(3) Finally, such treatment would, in many cases, be rendered much more effective if it were preceded by a period of continuous observation and diagnostic investigation in hospital. The limited facilities available to a travelling field clinic make anything in the nature of elaborate investigation impossible. It has become evident that temporary admission to a special observation unit would assist materially in the proper treatment of many of these cases.

In order that such facilities may be available to the clinics, the Province now has under construction a separate hospital unit especially designed and equipped for the diagnostic study of children suffering from convulsive disorders. Special care has been taken in the planning of this unit to include adequate facilities for the educational and recreational needs of this type of patient. It is believed that this special hospital provision will result not only in the more adequate diagnosis and treatment of convulsive disorders in their early stages, but will prove of particular assistance in the emotional and social re-adjustment of patients who present personality difficulties.

BIBLIOGRAPHY.

1. Pollock, H. M.: A statistical review of convulsive disorders in the United States. Am. J. Psychiat., 87: 665, Jan. 1931.
2. Thom, D. A.: Epilepsy and its rational extra-institutional treatment. Am. J. Psychiat., 87: 623, Jan. 1931.
3. Lunacy and mental deficiency: The 23d annual report of the Board of Control for the year 1936. Part I: 61. (Issued by H. M. Stationery Office, London, England.)
4. Anderson, C. L.: Epilepsy in the State of Michigan. Mental Hygiene, 20: 441, July 1936.

A STUDY OF PROLONGED COMA FOLLOWING INSULIN SHOCK.*

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Introduction.

Coma resulting from severe and prolonged hypoglycemia is ordinarily terminable rapidly by either oral or intravenous administration of a sugar solution.

Occasionally following the administration of glucose (or other sugars) patient may fail to regain consciousness in the ordinary short period of time and the coma may even deepen or death ensue. This state has been variously termed "protracted coma," "prolonged coma," "protracted hypoglycemia," etc. I have chosen the term "prolonged coma" as best descriptive of this condition and shall use it throughout this discussion.

The following is a fairly typical case of prolonged coma which terminated in recovery with marked improvement in the patient's mental state:

Patient C. C. Age 32. Diagnosis: Dementia præcox, paranoid type, duration 11 months.

On the day preceding the prolonged coma the patient exhibited late in the coma moderate respiratory distress of the asthmatic type. This subsided with the return to consciousness which was prompt after oral glucose. The prolonged coma occurred on the tenth day of the insulin treatment and followed the third coma.

The day of the prolonged coma patient received 240 units of insulin at 6.45 a. m. Mild restlessness began at 7.30, drowsiness and sweating at 8.30, light coma with the disappearance of restlessness at 9.30 and deep coma by 10 a. m. Shortly after this mild jactitation began. At 10.30 his breathing was stertorous and this gradually developed into moderately severe asthmatic dyspnœa. He was tubed at 10.45 and given 700 c.c. of 33.3 per cent glucose solution. He slowly displayed unorganized movements of the extremities, but there was no return to consciousness. The respiratory difficulty gradually became more marked until great muscular effort was required to breathe. He became slightly cyanotic although his pulse remained strong at rate of 100 per minute. He was given 1 c.c. of epinephrine hypodermically and

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100 c.c. of 50 per cent glucose solution intravenously. Following this patient became extremely restless, thrashing about in bed. By 11.45 there was an increase in respiratory difficulty and the motor excitement continued. In an effort to diminish the respiratory difficulty 1 c.c. of epinephrine was again injected hypodermically and 75 c.c. of 50 per cent glucose solution given intravenously. The respiratory difficulty and cyanosis increased. Since the epinephrine had not diminished the respiratory difficulty he was given 15 c.c. of paraldehyde by tube at 12.30 p. m. to test the sedative effect on the dyspnœa. By 1.15 p. m. he was still extremely restless. The respiratory difficulty and cyanosis had lessened only slightly. His stomach was drained by tube and the fluid smelled strongly of paraldehyde. The fluid removed from his stomach was in excess of the amount instilled and it was thought that very little of the paraldehyde had been absorbed. His stomach was thoroughly washed with an alkaline solution and he was given 500 c.c. of a regular egg, malt, milk tube feeding and another 8 c.c. of paraldehyde. During the next two hours the restlessness gradually lessened and respiration gradually became deep and easy. At 4 p. m. he was still in deep coma with areflexia and failure to respond to painful stimuli. His blood pressure fell from 160/100 to 90/50 and his pulse became rapid and weak. He was given large doses of digitalis and 100 c.c. of 50 per cent glucose solution were given intravenously on two occasions during the afternoon and evening. His temperature rose to 103.8° rectally and he continued to have many râles and wheezes in his lungs despite the now apparently effortless respiration. He continued in extremely deep coma with areflexia for 48 hours and had to be catherized and tubefed. By this time all paraldehyde odor was gone and this drug could be excluded as a basis for his deep coma. For the next 24 hours he remained in deep coma. After 72 hours of coma he began to move, attempted to swallow and began to mumble a little. He gradually regained strength and was in a semi-conscious state during the next five days. His temperature had returned to normal. For the next five days he was somewhat confused, slow of speech, and awkward in performing voluntary movements. During the next five days there ensued a marked improvement in patient's mental state. No improvement had been noticed prior to the prolonged coma. Symptoms of the "organic reaction type" of mental clouding disappeared and patient became friendly, cooperative and readily communicative. He still showed queer behavior and thought he could control the landing of the airplanes at a nearby airport. He was started on insulin again 15 days after recovery from the prolonged coma. Whereas the shock dose prior to the prolonged coma had been 240 units, thereafter 50 units were sufficient to produce shock. Deep quiet coma was achieved without complications for a total of 23 shocks. At the end of the treatment he had good insight, normal emotional response, no delusions, expressed ambition and exhibited a spirit of friendliness and cooperation. Early details of his illness were easily recalled and readily discussed but his memory for later details was hazy. It has now been five months since discharge from the sanitarium and patient is "more normal than ever before and has improved since he left the sanitarium," according to word received from his wife.

There are differences of opinion as to what should be called a prolonged coma. In order that a comprehensive idea may be obtained as to what physicians using insulin therapy have considered a prolonged coma, every case on which the information asked for has been given has been included in this report. Adopting this comprehensive idea as a standard, I have not included as prolonged coma those rather frequent cases which fail to respond to the oral administration of glucose solution but which do respond promptly to the intravenous injection of glucose. Nor have I included the protracted shock described by W. Kraulis [1] which is produced voluntarily and is merely a prolongation of the ordinary insulin coma, with the administration of just enough glucose from time to time to prevent irreversible nervous tissue changes.

Those cases which evidently a few physicians (although none who contributed to this study) regularly classify as prolonged coma, will be described briefly. After more prolonged or deeper coma than usual the patient is tubed and thereafter responds very slowly. Perhaps at the end of one-half hour some voluntary movements and the ability to appreciate sensory stimuli have returned but the patient remains in a semi-stuporous condition. He is then usually given variable amounts of intravenous glucose and other medication. Following this treatment there is a more rapid return to his usual mental state but the recovery period is still longer than usual, so that one to three hours may elapse before the patient is sufficiently awake to comprehend his situation or to perform voluntary movements, such as walking or shaving.

The difference between these cases and those of prolonged coma discussed herein is easily seen. In the cases just described there is a slow steady return of nervous tissue function and this return of function can be seen very shortly after the glucose administration. These cases do not show alarming symptoms. The difference between this state and the usual response to glucose is merely one of time. Many investigators have spoken of these patients, not as showing prolonged coma, but as being "habitually slow" in responding.

It is possible there are statistical errors in this report. At least two institutions responded only with the information that they had seen "several" or "many" cases of prolonged coma, "enough in fact to warrant a paper on the subject." This would indicate

that the frequency of prolonged coma in these institutions will be much higher than the incidence given herein.

In an attempt to minimize possible error, questionnaires were sent to many institutions where I had heard that one or more cases of prolonged coma had been observed. Several institutions returned only the information that they had seen no cases of prolonged coma in their insulin therapy, without giving the total number of completed insulin cases. Of course, these could not be included in this report and the two opposites should tend to lessen the statistical errors.

Although prolonged coma differs in many ways from ordinary hypoglycemic coma some observers [2] do not consider it a complication but merely an insulin therapy "reaction type." To these authors as well as to others [1] prolonged coma is a desirable type of insulin reaction and one usually followed by marked improvement in the patient's mental state. To still others [3] with equal experience with the therapy prolonged coma is a definite complication and something to be avoided.

It was due to these apparently conflicting opinions that I undertook a study of the prolonged coma as seen in the United States. A detailed questionnaire was sent to geographically representative institutions. In all, 20 institutions replied with the complete information sought, representing a total of 1173 completed insulin cases.

Each institution's completed cases together with the corresponding number of prolonged comas is listed below.

Completed cases:

70 10 95 23 30 24 41 36 69 70 40 108 58 57 35 20
114 96 27 150

Prolonged comas:

1 1 3 2 0 1 1 3 0 2 0 0 0 2 0 1 1 0 1 6

Twelve institutions replied with incomplete data. The total figures as given by three of them are reported below.

Completed cases:

87 150 200

Prolonged comas:

1 "1 or 2" "about 50"

At least two of these 12 will have reported their group of prolonged comas elsewhere.

Description of the Prolonged Coma.

Prolonged coma is easily recognized. After the ordinary morning of hypoglycemia and coma or after a hypoglycemia which was marked by unusual reactions on the part of the patient (such as an erythematous rash, severe motor phenomena, respiratory disturbance, cardiovascular failure or a very deep, quiet coma) the administration of the initial glucose is not followed by the usual return to consciousness. Instead the patient continues in coma.

If unusual reactions have not occurred before the initial glucose administration they usually appear within an hour or two thereafter, and consist usually of fever, diarrhœa, vomiting, respiratory disturbances either in the form of "asthmatic type" of dyspnœa, apnœa, for long periods with resulting cyanosis, or irregular breathing of the Cheyne-Stokes type, cardiovascular disturbances such as weak rapid pulse with or without irregularities, a fall in blood pressure to surgical shock levels, extreme restlessness or deep, quiet coma. Gradually failing respiratory or cardiac action may terminate the patient's life.

Any or all of these pathologic signs may be seen in the course of an individual prolonged coma. A patient frequently shows signs of disturbance of first one and then the other vital center (or body system).

If prolonged coma ends in recovery, this recovery is not sudden as is the recovery from the ordinary hypoglycemic coma but very gradual with the recovery of first one and then another nervous system function. Even after the acute symptoms have passed and the patient has apparently recovered from the coma itself a clouded mental state of the "organic type" with difficulty in concentrating, confusion, disorientation, memory impairment, etc., may persist for as long as two to three weeks.

Statistics.

Of the 25 instances of prolonged coma reported in our study four ended fatally. This suggests that prolonged coma has a mortality rate of 16 per cent while the death rate due to this complication in the total insulin therapies reported is 0.33 per cent. If we assume that 40 shocks per patient is the average extent of an insulin therapy our study would represent 46,920 shocks and

we may then expect prolonged coma to ensue once in every 1877 instances of coma.

Sakel has said in his early publications that deep protracted shock is preferable in the acute paranoid schizophrenias and later [4] has recommended such effect in all chronic cases. Easton [5] and Rossman and Cline [2] have reported immediate improvement in the patient's mental state following recovery from prolonged coma. So impressed was W. Kraulis [1] with the benefits of an unintentionally produced prolonged coma that he sought a method of producing it at will.

In this study of 25 prolonged comas, unintentionally produced, seven showed marked to moderate improvement, two showed slight improvement, while 12 showed no improvement in the mental state following recovery from the coma. Of the seven who showed improvement, two lost that improvement (relapse) within a few days.

With the exception of the fatalities only one case was thought to show any persistent evidence of brain damage (*i. e.*, after two to three weeks). The occurrence of prolonged coma does not contraindicate further insulin treatment as 12 cases were given further shocks, as high as 45 more, without the occurrence of persistent complications. It should be noted that in several cases no further insulin therapy was attempted after recovery from prolonged coma.

No significant conclusions could be drawn from a study of the age, sex, type of illness, or duration of illness of these patients. The earliest insulin injection to be followed by prolonged coma was the fifth, while the corresponding coma was the second. However, prolonged coma may appear first after 37 uncomplicated comas. The average number of comas preceding prolonged coma was 14.

The shortest duration of prolonged coma in this study was three hours. The longest prolonged coma reported which ended in recovery lasted 17 days. The average duration was 60.9 hours.

It apparently makes no difference whether the glucose used to terminate the ordinary coma is given intravenously or by gavage insofar as the production of prolonged coma is concerned. Fifteen patients received their initial glucose by gavage, eight by the intravenous route, and two by both routes simultaneously. Eleven

patients had shown some "unusual" reaction to the insulin on previous injections, either as extreme excitement, asthmatic type of respiratory distress, severe motor phenomena or in being "habitually slow" to respond following the administration of the glucose solution. At least nine patients showed similar "unusual" reactions plus erythematous rash, convulsion or cardiovascular collapse previous to the administration of the initial glucose on the day the prolonged coma ensued.

In all cases, except one, glucose was administered frequently enough during the prolonged coma to render the blood sugar normal or in excess of normal in every case in which it was taken. Persistent hypoglycemia is therefore not a cause of the prolonged coma.

The shock dose of insulin for the individual patient frequently changed following a prolonged coma but the average in this series remained almost constant, some patients requiring a great deal more insulin while others required much less to produce further shock.

As it has been suggested that the longer a patient is left in coma or hypoglycemia the more likely will prolonged coma ensue, I list below the total time from the insulin injection to the first glucose administration together with the corresponding durations of coma before the glucose was given in those cases in which such information was given. Time is in hours.

Injection of sugar:

5 3¾ 4¼ 4½ 4⅚ 4 3 4 5 4 3 3 5 4½ 6½ 5½ 7¼ 4 7

Coma to sugar:

1 ½ 2¼ 1 1½ 1½ 1½ 2 ¾ 1½ 2 1½ 1¼ 2 2 3 2¼ 1¼ 3

Injection of sugar:

6⅓ 2½ 4

Coma to sugar:

2 1½ 1

This makes the average time of potential hypoglycemia 4.47 hours and the average duration of coma prior to the initial glucose administration 1.58 hours.

Most of these cases of prolonged coma exhibited abnormal physical signs. Fever and tachycardia were almost invariably present, the fever reaching as high as 106 degrees. Diarrhea and

vomiting were present in about one-half the number. Other abnormal signs were convulsions, apnœa, dyspnœa and cardiovascular collapse.

Treatment.

The methods used by contributors to this series to combat the prolonged coma and to sustain life have been mostly symptomatic. Repeated administration of intravenous glucose solution was used in almost every case. Epinephrine was used in over half the cases. Other drugs used frequently were coramine, extract of the suprarenal cortex, phenobarbital, atropine, digitalis, caffeine, insulin, normal saline solution, morphine, paraldehyde and vitamin B_1. Vitamin B_1 has been widely used and some authors [2, 6] feel that it is the most efficacious remedy. Intravenous calcium, lumbar puncture, oxygen or carbogen inhalations or various stronger sedatives for the marked excitements have all been recommended. The frequent slow intravenous administration of 10 per cent glucose in normal saline solution was reported more efficacious than the more rapid injection of more concentrated solutions.

The most rational procedure appears to be to administer intravenous glucose frequently enough to maintain the blood sugar at normal to slightly above normal together with vitamin B_1 by the same route. Other medications will depend upon the predominant symptoms at the time. The extreme restless states require fairly heavy sedation. A type of medication that does not depress the respiratory center is preferable. To aid the heart digitalis, coramine or caffeine may be used. To stimulate the failing respirations lobeline, metrazol and caffeine are valuable. Artificial respiration should be resorted to if necessary. The severe asthmatic type of respiratory distress with cyanosis may become a difficult problem and in my case the administration of epinephrine gave no relief. Epinephrine must be used with caution in prolonged coma, due to its tendency to increase restlessness. Several contributors report the onset of convulsions following the injection of adrenalin in an attempt to combat the coma. There are certainly points in favor of the use of small doses of insulin along with the intravenous glucose, of suprarenal cortex preparations, of intravenous calcium, of lumbar puncture, and of inhalations of oxygen. If frenzied

excitement occurs methods must be undertaken to prevent the patient from injuring himself. A relative alkalosis may develop, due to hyperventilation. Prolonged coma with persistent hyperventilation particularly when associated with tetany should be treated with carbon dioxide inhalations or rebreathing. The same condition may result from persistent vomiting during prolonged coma but must be treated a little differently, namely by saline injections.

If the coma is prolonged for days suitable nourishment must be given by tube. Good general nursing care is essential following the disappearance of the acute symptoms. Not much activity should be demanded of the patient and no further insulin shock should be attempted until the complete disappearance of the "organic reaction type" symptoms.

A thorough pathologic study should be undertaken in case of fatality.

Discussion.

We are all familiar without further description with the ordinary reaction to Sakel's insulin therapy and of the rapid return to normal of nervous system function following the administration of glucose. We know that the amount of available blood sugar necessary to keep the nerve cells viable and capable of an early return to normal functioning when glucose is once again available (reversible reaction) is much smaller than the amount of sugar necessary for the normal functioning of the nervous tissue.

There is no agreement as to whether or not prolonged hypoglycemia produces definite demonstrable nervous tissue damage. Conflicting viewpoints are expressed in some reports.[7, 8, 9, 10, 11] However, it is certain that death will result if hypoglycemia is maintained at a low enough level for a long enough time. From this fact and from the clinical neurologic signs it must be assumed that hypoglycemia impairs the normal function of the nervous tissue, as well as other tissues, and that if maintained long enough this disturbance of function is irreversible. That is, death ensues.

There are a degree and duration of hypoglycemia, undoubtedly varying somewhat for each individual, at which the reversible changes in nervous tissue function become irreversible. There is a borderline state between the two and the length of time a

patient may survive in this state depends on the individual constitution. In my estimation prolonged coma represents this borderline state.

The inability to utilize the available glucose, the continued clinical evidence of disturbance of function of the nervous tissue, the slow recovery with the appearance of an "organic reaction type" of mental clouding following the recovery from the acute phases of prolonged coma, would seem to show that the nervous tissue has approximated irreversible change.

From this study and from the opinions of those consulted it appears that prolonged coma is more apt to occur: (1) When the depth of ordinary coma is profound, (2) the longer the duration of hypoglycemia and/or coma, (3) when any "hypersensitivity" to insulin exists (as shown by other evidence of hypersensitivity such as urticaria or asthmatic type of respiratory distress), (4) when a deficiency of tissue essentials such as vitamin B exists (assumed from the beneficial effect of their administration), (5) as suggested by several contributors when the dosage of insulin is increased too rapidly. (This is a doubtful factor as shown by the following: In approximately one-half of the reported cases the prolonged coma ensued on either the second, third or fourth coma. In the other half of the cases the prolonged coma occurred, as an average, on the 20th coma. In the former the rate of increase of insulin dosage averaged 12.5 units per day and in the latter averaged 10.6 units per day), and lastly, (6) it appears to be due, as explained, to the fact that a certain number of patients possess a constitution which is unable to stand with average impunity a prolonged period of hypoglycemia.

Conclusions.

It appears from this study that:

1. Prolonged coma occurring in the course of insulin therapy is a definite complication and one which should be avoided whenever possible.

2. Prolonged coma is not usually followed by improvement in the patient's mental state.

3. In a few cases dramatic improvement in the patient's mental condition may be noted upon recovery from prolonged coma.

4. Such improvement as may be obtained in the minority of cases does not justify attempts to produce prolonged coma as the mortality rate is too high, 16 per cent in this series.

5. Prolonged coma may be expected to occur once to every 1877 ordinary comas.

6. Treatment, possible causes, and possible errors in these statistics have been discussed.

I wish to thank the many contributors who have made possible this study.

BIBLIOGRAPHY.

1. Kraulis, W.: Protracted shock in the treatment of schizophrenia and results of the therapy in Riga, Latvia. Am. J. Psychiat.—Supplement, 94: 324, 1938.
2. Rossman, I. Murray, and Cline, William B., Jr.: The pharmacological shock treatment of chronic schizophrenia. Am. J. Psychiat., 94: 1323, 1938.
3. Dussik, Karl Theo.: Three and a half years of the hypoglycemic therapy of schizophrenia. Am. J. Psychiat.—Supplement, 94: 269, 1938.
4. Sakel, M.: On the reports of Maier, Klaesi and Wilmanns. Am. J. Psychiat.—Supplement, 94: 341, 1938.
5. Easton, Norman L.: A case of protracted hypoglycemia. Am. J. Psychiat., 94: 853, 1938.
6. Demole, V.: Dementia præcox, hypoglycemia, insulin, vitamin B_1. Am. J. Psychiat.—Supplement, 94: 59, 1938.
7. Baker, A. B., and Lufkin, N. H.: Cerebral lesions in hypoglycemia. Archives of Pathology, 23: 190, 1937.
8. Grayzel, David M.: Changes in the central nervous system resulting from convulsions due to hyperinsulinism. Archives of Internal Medicine, 54: 694, 1934.
9. Smith, Margaret G., and Seibel, Marshall G.: Tumors of the islands of langerhans and hypoglycemia. Am. J. Pathology, 7: 723, 1931.
10. Accornero, F.: Experimental histopathological researches on insulin shock. Am. J. Psychiat.—Supplement, 94: 131, 1938.
11. Nicolajev, Vladimir: A special glial alteration after repeated insulin shock in animals. Am. J. Psychiat.—Supplement, 94: 302, 1938.

RADIOLOGIC GASTROINTESTINAL STUDIES IN EPILEPSY.

By LEON J. ROBINSON, M. D.*

In epileptic patients the relationship of gastrointestinal abnormalities to the fundamental nature of epilepsy has been analyzed by several observers. Out of these reports have emerged conflicting opinions upon the incidence of gastrointestinal pathology among patients with epilepsy. Reed [1] actually considered epilepsy a symptom of splanchnoptosis. He concluded that all epileptics show splanchnoptosis, an opinion he based upon 810 consecutive examinations. In an earlier report, this same author [2] stated that epileptic patients who were operated upon revealed dilatation of the duodenum and enlarged retroperitoneal glands. Bethea's gastrointestinal study of 400 epileptic patients demonstrated an amazingly large number of abnormalities, only 21 patients having normal stomach findings.[3] In contrast to Reed's [1] findings of constant splanchnoptosis, Bethea noted colonic ptosis in only 4 per cent of his patients. Harryman and Donaldson [4] observed fewer and less startling gastrointestinal abnormalities. Of their 50 patients 25 showed hypermotility, usually of the colon, 19 slight stasis, and only one showed gastrointestinal ptosis. At autopsy Caro [5] found the same percentage (18 per cent) of intestinal adhesions and peritoneal bands in 280 patients with epilepsy as was found in 755 patients without this disease. He believes that most of the adhesions are the result, rather than the cause, of stasis.

Lennox and Cobb's tabulated data of 423 autopsies at Craig Colony [6] failed to show any greater degree of organic gastrointestinal abnormality in epileptic patients than in non-epileptic institutional patients.

Several observers have noted that, at times, the correction of gastrointestinal abnormalities in patients with epilepsy has eliminated further seizures. Robertson [7] stated that of 500 patients with chronic intestinal stasis 27 had convulsions. Fifteen of these 27

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patients received adequate treatment for the intestinal stasis with the result that five patients were freed of seizures and all but four were improved. Barclay [8] and Block [9] each reported a single case of this kind. Acidophilus milk was fed to 12 patients daily by Kopeloff, Lonergan and Beerman [10] with reduction of seizures in four of the 12 patients. They also gave kaolin to another group but without benefit.

Lennox and Brown [6] have seen complete cessation of seizures following orthopedic treatment in three patients with poor posture and x-ray evidence of ptosis of the stomach and transverse colon. Axtell [11] found angulation and fecal impaction of the sigmoid in 45 patients, eight of whom became free of seizures following medical treatment. Reed [12, 13] stated that many cases were relieved following removal of a section of cecum or colon. Armstrong [14] reported that colectomy stopped attacks in two patients. On the other hand, Brewster [15] failed to note any improvement from colectomy in 12 patients.

Constipation is commonly said to be present in epileptic patients, but as Lennox and Cobb [6] point out, even this simple statement lacks adequate proof. Nielson [16] found that there was a higher percentage of constipation among 200 epileptic patients than among 200 non-epileptic persons.

Weingrow [17] found constant gastrointestinal changes in cats in whom convulsions were experimentally induced by absinth. Dilatation of the stomach was the most prominent finding, dilatation of the colon was next in frequency, and contraction of the colon was third in frequency.

In studying the influence of the cerebral cortex on gastrointestinal movements, Watts and Fulton [18] found that in monkeys, faradic stimulation of many portions of the premotor area (especially bordering on the superior precentral sulcus) produced vigorous intestinal movements. The latter were usually initiated in the cecum and lower part of the small intestine, but occasionally were initiated in the colon. Often, too, there resulted transient, non-obstructive intussusceptions.

In studying the cerebral localization of epileptic manifestations, Penfield and Gage [19] noted that an aura of pain or epigastric distress may arise from activity of the cerebral cortex. In conscious patients under local anesthesia, they observed that stimulation of

area 7a caused pain in the right lower abdominal quadrant in one patient, and stimulation of the same area in another patient provoked an epileptic attack preceded by the usual epigastric aura.

In a large group of epileptic patients it is not unusual to find an occasional patient in whom visceral manifestations such as belching, nausea, epigastric distress and vomiting occur in place of the convulsive seizure. Several years later seizures may for the first time begin to accompany these phenomena. Levy and Patrick [20] found that among 500 patients with essential epilepsy 13 had such recurrent attacks to which in the course of time epileptic features were added. Watts and Frazier [21] reported two similar cases. They postulated motor autonomic representation for the gastrointestinal tract in the cortex of man. Watts [22] states that it is probable that nausea, epigastric distress and possibly vomiting may be the result of neuronal discharge from the motor autonomic portion of the cerebral cortex. He also believes that "the epigastric aura and other abdominal sensations usually are not 'referred' sensations but rather the result of vigorous, and probably abnormal, movements of the gastrointestinal tract."

Because of these many suggestive gastrointestinal findings in patients with epilepsy, the present investigation was carried out. A roentgenographic study of the gastrointestinal tract was made in each of 100 patients with epilepsy. For the most part they were patients with idiopathic epilepsy. The single method barium sulfate contrast medium was used. Roentgen examination was made at once, and at 6, 24 and 48 hours, and in many cases at 72 hours as well. Results are tabulated in Tables I and II.

Eighty-six of the 100 patients had a normal gastrointestinal series. Of the 14 patients with abnormalities four had a duodenal ulcer, one a gastric ulcer, three showed six-hour gastric retention (none of whom showed ulcers), one adhesions about the ileum, two had diverticuli of the colon, and three had a spastic colon. In two of the latter, hyperperistalsis was evident. Of the three patients with six-hour gastric retention one showed 30 per cent retention and the other two 20 per cent and 15 per cent, respectively.

Of the entire 100 patients, 23 had some type of gastrointestinal aura. It is interesting to note that only three of these patients with such aura had gastrointestinal abnormalities. Two of these patients had a duodenal ulcer while the third had colonic diverticuli. It is

also to be noted that the presence of one or the other of these lesions in four patients was unaccompanied by any gastrointestinal aura. Furthermore, the remaining seven patients with gastrointestinal abnormalities had no gastrointestinal aura. Of these

TABLE I.

Total No. G.I. Series.	No. Normal.	Total No. Abnormal.
100	86	14

TYPE OF ABNORMALITIES AND NUMBER.

Duodenal ulcer	4
Gastric ulcer	1
6-hour gastric retention	15 per cent.... 1; 20 per cent.... 1; 30 per cent.... 1 — 3
Adhesions of ileum	1
Colonic diverticuli	2
Spastic colon	3 (2 showed hyperperistalsis).

PATIENTS WITH G.I. AURA.

Total Number 23.

TYPE OF G.I. AURA AND NUMBER.

Epigastric distress.	Peculiar sensation in epigastrium or esophagus.	Hypogastric pain and vomiting.	R. L. Q. pain (until appendectomy).	Nausea.
6	4	1	1	1

Vomiting.	Nausea and vomiting.	Eructation of gas.	Borborygmus.
3 (one with spell)	2 (one ½ hr. before)	4	1

latter seven patients, three manifested gastric retention of 15 per cent to 30 per cent, three showed spasticity of the colon, and one showed adhesions about the ileum. Hence, of 14 patients with gastrointestinal abnormalities, only three possessed gastrointestinal auræ.

Of the five patients noted by x-ray to have either gastric or duodenal ulcer, gastrointestinal symptoms were present in all. Four

TABLE II.

COMPARISON OF THE ABNORMAL G.I. SERIES FINDINGS IN 14 PATIENTS WITH G.I. SYMPTOMS OR AURA.

Nature of G.I. abnormality.	G.I. aura.	G.I. symptoms not related to seizures.
Duodenal ulcer	0	Epigastric distress 2 hours after meals.
Duodenal ulcer	0	Epigastric distress 1½-3 hours after meals.
Gastric ulcer	0	Epigastric distress ½ hour after meals.
Duodenal ulcer	Eructation of gas just before seizures, and vomits during seizures.	Periodic epigastric pain and vomiting at times at night.
Duodenal ulcer	Epigastric pain and vomiting 2-3 hours before seizures.	Epigastric pain and vomiting 2-3 hours after meals.
Six-hour gastric retention 30 per cent.....	0	0
Six-hour gastric retention 20 per cent.....	0	Occasional epigastric pain one hour after meals.
Six-hour gastric retention 15 per cent.....	0	0
Adhesions about ileum...	0 (vertigo before spell)	0
Colonic diverticuli	0	Epigastric distress following fatty foods.
Colonic diverticuli	Shaking epigastric sensation ½ hour before seizure.	0
Spastic colon	0	0
Spastic colon	0	0
Spastic colon	0	0

All patients but one with G.I. pathology have idiopathic epilepsy.
One patient with colonic diverticuli has epilepsy symptomatic of agenesis.

of these complained of typical ulcer pain or distress occurring from one half hour to three hours after meals, while a fifth patient had periodic epigastric pain and vomiting at night. The

latter patient had a gastrointestinal aura consisting of eructation of gas just before seizures, and he vomited during seizures. One of the patients with duodenal ulcer who had typical epigastric pain and vomiting two to three hours after meals, had both of these features as an aura two to three hours before seizures. One patient with colonic diverticuli had a "shaking sensation" in the epigastrium half hour before seizures, but no symptoms at other times. Another patient with colonic diverticuli had epigastric distress following fatty foods but had no gastrointestinal auræ. One patient with 20 per cent gastric retention at six hours had occasional epigastric pains one hour after meals but no gastrointestinal aura. Neither the patient with adhesions about the ileum nor the three patients with spastic colon had gastrointestinal aura or gastrointestinal symptoms at any time.

Of the 14 patients showing gastrointestinal abnormalities, all but one had idiopathic epilepsy. The latter's epilepsy was symptomatic of agenesis (diagnosis being sustained at autopsy) and the gastrointestinal pathology consisted of colonic diverticuli. He had no gastrointestinal aura.

As has been pointed out, 23 of the 100 patients had some form of gastrointestinal aura. The types of auræ were varied and represented the common varieties of gastrointestinal symptomatology. These included epigastric or abdominal distress or pain, nausea, vomiting and eructation of gas. The time interval between gastrointestinal auræ and seizures varied from a few seconds to three hours. As a rule the more definite the gastrointestinal aura the longer was the interval warning before a seizure.

We concur with Watts [22] who believes that "the epigastric aura and other abdominal sensations usually are not 'referred' sensations but rather the result of vigorous, and probably abnormal movements of the gastrointestinal tract."

Summary and Conclusions.

Gastrointestinal series were carried out in a group of 100 patients with epilepsy. Eighty-six patients had normal gastrointestinal findings. Fourteen patients showed various abnormalities but these showed no uniformity. It was concluded that there is no characteristic gastrointestinal pathology in epilepsy.

Twenty-three of the 100 patients examined had some type of gastrointestinal aura. Only three of these had any gastrointestinal abnormality. It was concluded that epileptic gastrointestinal auræ do not depend on demonstrable gastrointestinal abnormalities.

BIBLIOGRAPHY.

1. Reed, C. A. L.: Epilepsy, a symptom of splanchnoptosis. Pennsylvania M. J., 24: 322, Feb. 1921. *Ibid.*, W. Virginia M. J., 15: 266, Jan. 1921.
2. Reed, C. A. L.: Diagnostic methods and pathological constants in idiopathic epilepsy (second report) radiographs. J. A. M. A., 66: 336-345, Jan. 29, 1916.
3. Bethea, W. R.: Gastrointestinal study of 400 epileptics: Preliminary report, South M. J., 20: 430-436, June, 1927.
4. Harryman, M. W., and Donaldson, S. W.: Radiologic gastrointestinal studies in epilepsy. J. A. M. A., 81: 813-815, Sept. 8, 1923.
5. Caro, H.: Intestinal adhesions and peritoneal bands in epileptics. Boston M. and S. J., 177: 208-210, Aug. 16, 1917. *Ibid.*, Incidence of intestinal adhesions as factor in chronic intestinal stasis in epilepsies. Boston M. and S. J., 176: 697-699, May 17, 1917.
6. Lennox, W. G., and Cobb, S.: Epilepsy, p. 59-62. Williams and Wilkins Co., 1928.
7. Robertson, A. W.: Chronic intestinal stasis and epilepsy. Brit. M. J., 2: 1191-1193, Dec. 27, 1924.
8. Barclay, H.: Relation of epileptic syndrome to intestinal toxemia. Am. Med. (Burlington), 23: 242, April, 1917.
9. Block, E. B.: Epilepsy: Case with megacolon and polymastia. South M. J., 16: 15-17, Jan., 1923.
10. Kopeloff, N., Lonergan, M. P., and Beerman, P.: L. acidophilus in epilepsy. Quoted by Lennox and Cobb (6).
11. Axtell, W. H.: Acute angulation and flexure of the sigmoid as a causative factor in epilepsy. Virginia M. Semi-monthly; Richmond, 21: 409, 1916. Quoted by Lennox and Cobb (6).
12. Reed, C. A. L.: Diagnostic methods and pathological constants in idiopathic epilepsy. J. A. M. A., 66: 336-345, Jan. 29, 1916.
13. Reed, C. A. L.: Typical recoveries in Iowa from chronic convulsive toxemia (epilepsy) following surgical correction of abdominal viscera. J. Iowa M. Soc., 10: 204-208, July, 1920.
14. Armstrong, G. E.: Relation of cecum and ascending colon content to idiopathic epilepsy. Practitioner (London), 117: 288-291, Nov. 1926.
15. Brewster, Colectomy in treatment of epilepsy. Boston M. & S. J., 186: 482, April 6, 1922. Quoted by Lennox and Cobb (6).
16. Nielson, J. M.: Etiology of epileptiform seizures. Med. J. & Rec. Supp., 122: 338-340, Sept. 16, 1925.

17. Weingrow, S. M.: Visceral conditions in general epilepsy and in conditions of experimental origin. Am. J. Psychiat., 88: 737-746, Jan. 1932.
18. Watts, J. W., and Fulton, J. F.: Intussusception: The relation of the cerebral cortex to intestinal motility in the monkey. New England J. Med., 210: 883-896, April 26, 1934.
19. Penfield, W., and Gage, L.: Cerebral localization of epileptic manifestations. Arch. Neurol. and Psychiat., 30: 709-727, Oct. 1933.
20. Levy, D. M., and Patrick, H. T.: Recurrent "attacks" other than migraine and infantile convulsions preceding "true" epilepsy. Arch. Neurol. & Psychiat., 20: 443-463, Sept. 1928.
21. Watts, J. W., and Frazier, C. H.: Cortical autonomic epilepsy. J. Nerv. & Ment. Dis., 81: 168-175, Feb. 1935.
22. Watts, J. W.: The influence of the cerebral cortex on gastrointestinal movements. J. A. M. A., 104: 355-357, Feb. 2, 1935.

ADMINISTRATIVE CONDITIONS AND PROBLEMS OF THE CALIFORNIA MENTAL HOSPITALS.*

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In presenting this paper on the administrative conditions and problems of the mental hospitals of California to a group of scientific and professional people, I am mindful of that old injunction that "fools rush in where angels fear to tread." Because I am a layman, I feel highly honored in being asked to address you and necessarily my remarks will be confined to the layman's viewpoint.

California started to function as a state when its first legislature met in 1849, but several years before that it was attempting to meet the mental health problem of its early residents in its few outposts of civilization. From early records we find that in 1849 mental patients were being cared for in an old sailing vessel anchored in San Francisco Bay. The great influx of gold seekers in 1849, attended by mental breakdowns due to the trials and tribulations, disappointments and excitements, presented a problem which the old sailing vessel could not handle because of its limited capacity, and from that day California has had the problem of over-population in its mental hospitals.

The first mental hospital was opened at Stockton in August, 1851, two years after the first legislature met, and since that time six other mental hospitals have been built, the newest one partially completed and partially occupied within the last year at Camarillo, in Southern California.

Twenty years after Stockton State Hospital was opened, its population had increased to 1090. Today the population in the seven mental hospitals of this state is 21,689. This is an immense population when it is considered that it has accrued over a period of 89 years.

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Our state, like many other states, has not kept up its building program to meet the demands for the care of the mentally sick. This has resulted in a continuous problem of providing adequate facilities for them. Demands of other departments of the state government have been given first consideration, either because of political emoluments or because of a more vociferous clamor for relief. In 1934 the population of our mental hospitals was 18,640 and our bed capacity was 14,322, making an over-population of 4318, or 30.15 per cent. We reached our highest peak of over-population in July of 1936 when we had 20,248 patients. Our bed capacity was 14,433, giving us an excess of 5815, or 40.29 per cent.

In a survey of the mental hospitals in 1934 it was found that attics, basements, occupational therapy rooms, temporary structures, day rooms and halls were converted into temporary wards. Floor beds were in common usage and our staffs were meeting the problem as best they could under these most disadvantageous conditions. This survey revealed that it would take approximately twenty million dollars to bring the capacity up to the normal demand. The state government in 1934 was facing a thirty million dollar deficit. The present governor had been elected on a platform of balancing the state budget and it was therefore impossible to obtain sufficient money to build up the mental hospitals to normal capacity. A bond issue for institution building was proposed to the people of the state but it was voted down because the politicians had tagged on to the appropriation bill several other desirable but unnecessary building projects. However, this department was successful in obtaining a four million dollar legislative appropriation in 1935 due to the publicity and interest that had been aroused in the state institutions. That four million dollar program has been completed and provided 2962 new beds, but the problem still exists. California's average annual net increase in mental hospital population is 800 patients so that in a four-year period enough population accumulates for a new institution.

In the 1937 session of the legislature, appropriations for six million dollars additional for new buildings were secured. This amount was amplified by a P. W. A. grant from the Federal Government of $2,016,164, making a total appropriation, which is now being expended, of $8,016,164. This will provide 4222 new beds. Total appropriations for new building purposes during the

two biennial periods total approximately 12 million dollars and provide 7184 new beds. When the building program is complete, this state will still have a problem of over-population although it will be cut down to less than 17 per cent. It is not necessary for me to tell this group the problems resulting from over-population and I know that our doctors and their staffs have your sympathy in meeting the condition. I cannot pass this point without paying my tribute to the superintendents and psychiatrists in our department for their loyal attitude and cooperative spirit shown during this trying period.

California's present method of operating its state departments is through a cabinet form of government. The principal departments are presided over by a director who holds appointment at the pleasure of the governor. These directors form the cabinet and the Department of Institutions is in that category.

Besides the seven mental hospitals, the Department of Institutions includes two homes for the feeble minded, three correctional schools, three institutions for the blind, a State Narcotic Hospital and the Bureau of Juvenile Research. The total population of all institutions in this Department is 26,866 and the number of employees is 3655.

All positions in this Department, with the exception of the director and one exempt position, are under civil service due to a constitutional amendment which was adopted by the people in 1934. Civil service is strictly adhered to and before an employee can be hired, he must have passed a written and oral examination and must have been placed in a favorable position upon an eligible list. This applies to medical superintendents and other institution superintendents, as well as to the lowest paid employee in the Department.

The director of the Department of Institutions appoints the medical superintendents from a civil service eligible list and the director has delegated his authority of other institutional employment to the medical superintendents, holding them responsible for their staffs and personnel.

Provision is made in this civil service law for emergency and temporary employees but they are confined to the strictest interpretation of emergency and temporary. An emergency employee cannot serve for more than 15 days and a temporary employee

cannot serve for more than six months and temporary employees cannot be appointed if eligible lists are available. This civil service legislation has its advantages and disadvantages, but to date we firmly believe that the advantages far out-balance the disadvantages.

During the past three years we have given considerable attention to personnel, in addition to improving conditions for patients. We have strengthened the requirements that must be met by those who seek employment as attendants, by requiring a high school education, practical nursing experience, the army standard for height and weight and by lowering the maximum age limit from 50 to 45.

Our employees were formerly paid on what we termed a net salary plan. To illustrate, attendants received a starting salary of $85 a month. $50 of that amount was paid in cash and $35 was rebated for maintenance which included room, meals and other services. We found considerable dissatisfaction which resulted in dissension and constant turn-over among this group of employees. In many cases employees were living off the grounds of the institution with their families and still were forced to pay $35 a month for services they did not receive. We changed this to what we term a gross salary plan. To illustrate this, an attendant receiving a starting salary of $85 a month is paid $85 a month and he pays back to the institution for those services which he receives. If he has a room on the grounds, he pays $11 a month for it. For each meal that he takes at the institution he pays $8 a month. Meals are handled through a method of meal tickets. The employee purchases the meal ticket from the secretary of the institution or other designated official and each time he takes a meal, the dining room supervisor punches his ticket. When this ticket is used up, he purchases another.

This system has presented an administrative financial problem. When the system was first put into effect we estimated that money for meal tickets and room services in the amount of $1,034,186 would be collected. Instead of that, approximately $526,189 will be collected this fiscal year and we are running $507,997 less in income than we had anticipated. Now that we have actual figures on how much this system is costing, we will be able to make ample provision for it in the future. The benefits to be

derived from the gross salary plan are intangible. We do know that we have built up the morale of our employees, and that we have eliminated much of the institutional gossip which originates with people living closely together and not having other interests. We know that our employees are becoming a part of the community adjacent to the institution. They are building homes and are paying taxes. They are renting homes, thus creating quite a problem in several of the institution communities, and are taking part in other community interests and activities.

We have already felt the effects of this plan in our building program. Previously, whenever we enlarged our institutions it was also necessary to enlarge our employees' quarters. We are eliminating many of these employees' quarters and have turned them into additional bed space for patients. While the cost of this plan at first blush appears very great, we feel that the benefits to the State government indirectly will be far greater than the additional cost. The gross salary plan does not apply to the executive staffs of the institutions because it has been felt that those holding certain executive positions should be available when duty calls at all times. A skeleton staff of other employees also resides on the grounds for emergency purposes.

The State Personnel Board, which has charge of civil service administration, has recently completed a salary survey which has again been of benefit to the employees. For example, the former salary range of an attendant was $85 to $105. The new salary range as a result of this survey is $100 to $140. Many other institution positions have been benefited proportionately; for example, our business managers who have had, under the direction of the medical superintendents, entire charge of the physical plants have been underpaid for the responsibilities that they carry. Through this survey they have been placed in a relative position slightly under the assistant medical director, with a salary range of $220 to $300, plus maintenance for self and family.

The cost of maintaining the seven mental institutions in California, in addition to outlay for construction, for the 1935-37 biennium was $8,509,496.71. This makes a per capita cost of $211.66 per annum. California's ratio of mental patients to population is slightly higher than the United States' average. The latest government figures, for the year 1935, show that we have 315 per

hundred thousand population while the United States' average is 269. This increase over the United States' average is probably accounted for by the fact that this state has had a large influx of residents of other states, both prior to and during the depression years. Of course this influx has increased during the depression years and this has materially affected our hospital population.

We use every possible means of cutting down our hospital population. At the present time we have 2589 patients on parole, most of them under the supervision of psychiatric social service workers. We have been attempting to increase the number of social service workers so that we may take care of more patients on parole. There is a decided economic advantage in this as we know that the normal case load of the social service worker is 100 patients. A social service worker's salary and expenses run approximately $2400 a year. At the present per capita cost, it would necessitate the sum of $21,166 to maintain these same one hundred patients in the institutions.

We return to their homes, cured or improved, 42 per cent of our hospital commitments. At the present time many of our social service workers are carrying a case load of 300 or more patients, which is too heavy a load for efficient work. We are using a method now of training our own social service workers as juniors, with the hope of increasing our senior staffs after the juniors have served for at least a year in the lower grade.

Because of this great influx of population to California, we also accumulate a large number of non-resident mental patients in our mental hospitals. Our law provides that we must return a patient to his home state if he has not resided in California for at least a year. This has necessitated the plan of special deport trains, which we have been running in the spring and fall of each year. We carry an average of 200 patients on each of these trains, together with attendants and doctors. This train runs as a special as far as Chicago. From there, two or three cars are placed on a regular train and carried through to New York. Most of our deport patients come from the central and southwestern states. We have reciprocal agreements with many states for the return of non-resident patients.

In our new building program we have adopted the one story, connected unit of construction. This spread-out plan is feasible in

California because of the peculiar climatic conditions which we have. All new buildings are of permanent construction, fireproof and earthquake proof. Our newest institution at Camarillo, which is now partially completed and partially occupied, will ultimately have a capacity of 6000 patients. We have completed the male unit, which has a capacity of 2400, and have at the present time 2000 patients in it. The female unit will have the same capacity and the hospital unit will have a capacity of 400 beds. Other special treatment buildings make up the balance. The type of architecture is Spanish and each ward has its own patio or exercise yard. A recreation center in this male unit occupies seven acres of ground, with ward buildings entirely surrounding it. We have used bright colors in furnishing and decorating it and it has all the appearance of a Spanish village with all wards connected. It is something entirely new in mental hospital construction and, although we have had patients in it for less than a year, we find that it is a very efficient type of construction for our climate and has great facility for administration.

Because of the location of our mental hospitals, which are mostly in agricultural or citrus centers, we carry on extensive farming operations. We are at present farming 8172 acres of land, in addition to operating poultry, dairy and hog plants. We also can most of our own fruits and vegetables. Last year our total farm revenue amounted to $1,129,406.00. Our total farm cost was $821,361.99, showing a net profit of $308,044.01 or 37½ per cent profit. All our products are sold by the farm to the institution at the prevailing wholesale price, and in addition we furnish occupational treatment for hundreds of patients who would otherwise have no exercise or interest.

This Department has in effect a food control system for all institutions. Through mathematical computation plus dietetic value, we are able to prepare attractive menus and at the same time figure with a fair degree of accuracy our needs for any given period. This department is presided over by a food administrator. The administrator prepares an estimate of the quantity of food for a given number of patients and furnishes this estimate to the supervising cook, who governs his menus accordingly. We have found that this system provides a varied and beneficial diet for our patients and has saved materially on our food costs.

Our meals are served at an average cost of 7.26¢ each in our hospitals. Menus are prepared for each week's service at each institution and sent to our head office. As an example of this food service a sample menu for one day's service consists of: breakfast: rolled oats, prunes, sugar, bread and butter, fried potatoes, coffee or milk; dinner: split pea soup, roast beef and gravy, squash, steamed potatoes, bread and butter; supper: carrot and raisin salad, macaroni and cheese, stewed tomatoes, bread and butter, indian pudding, tea or milk. Our basic menus were worked out under the supervision of competent dietitians and in conjunction with dietitians from the University of California.

The one thing that impresses me in this entire mental health program in California is the vast amount of money we are expending annually in caring for mental patients after they become ill and the small amount of money and effort that we are expending in preventing mental illness. It is true that we are carrying on some prevention and research work, particularly with insulin and metrazol and with alcoholic cases, but our facilities are limited and our staffs have been working under a heavy handicap due to our over-crowded condition.

With this thought in mind, we have proposed and are now formulating plans for two mental health centers in California, one to be established in the population center in the North and the other to be established in the population center in the South. These two mental health centers will have several functions. They will act as receiving units for all mental patients, where the patients will spend an observation period of not more than 30 days and will then be sent either to their homes, where they will be under the observation of trained psychiatric workers, or to the mental hospital best equipped to care for them. They will serve as diagnostic units and will be equipped for extensive research work. They will be used as training schools for attendants, nurses, staff doctors and university students. We propose ambulatory clinics where the public may obtain information and advice on mental health. Each of these institutions will be equipped with 400 beds and the population will be kept constantly moving so that 400 new patients can be cared for each month in each institution.

We feel that by the establishment of these two new units, still greater economy will result in the administration of our mental

hospitals by cutting down the number of commitments, by proper segregation of patients and through information that will lead to the prevention of mental disease.

In summing up my remarks I wish to point out the rapid growth of the mental hospitals in California, starting from nothing with the admission of California to statehood in 1849 to a population of 21,689 at present; the continuous problem of over-population, reaching its peak in July, 1936, with an over-crowding of 40.29 per cent; our efforts to meet this over-crowding by the expenditure of 12 million dollars in new building construction which will reduce this over-population to less than 17 per cent. California institutions are governed by a rigid civil service law in the selection of personnel. In addition to providing better and greater facilities for our patients we have made forward strides in improving the conditions of employment for our personnel. We have had splendid results in treating and curing our patients despite serious handicaps and I have outlined to you our proposal to improve and expand our receiving and preventive services.

DISCUSSION.

DR. FREDERICK W. PARSONS (New York, N. Y.).—To ask a man from New York State to talk about California is a great honor to the person invited to make the comparison. I am really surprised and gratified to learn of the excellent program and the progress which has been made in California within the last two years.

California is to be congratulated upon having a man of Mr. Lutgens' foresight, insight, interest and energy. If they are only wise enough to keep him sufficiently long, the 17 per cent overcrowding which, after all, is not desperate, will be further reduced.

Mr. Lutgens speaks of so many interesting things, it is hard to know where to begin. I am particularly struck, of course, with the civil service requirement recently introduced, and I hope that California will be wise enough to continue to adhere strictly to a civil service regimen.

Mr. Lutgens is perfectly conscious of, and says very truthfully, that there are disadvantages and advantages. He says something which others have known for a long while, and that is that the advantages greatly outweigh the disadvantages.

The only thing that Mr. Lutgens did not speak of is a shorter day. The state pays a generous wage scale, apparently has a very liberal food ration, but the per capita cost is so much less than New York's that I don't quite see where it comes in. It costs New York State about a dollar a day to feed, house and clothe a patient. Our wage scale is not as high as that of California.

We have recently had an eight-hour day, which brings the ward per capita down to one to six and a half; that is, one ward employee for every six and a half patients, and yet California operates its institutions at a $221-a-year basis, whereas New York State cost is $365.

Of course, there is a very great heating problem in New York which is not present here, but even that doesn't account for the $140 difference in the per capita cost.

I think many of us will be surprised that California finds a need for a six-thousand-patient hospital. That is a large institution anywhere. We in New York are not disposed to consider a six-thousand-patient hospital too large. As a matter of fact, we have several, and I think, as with civil service, there are advantages and disadvantages in a large hospital.

A state like California, rich, strong, lusty, growing all the time, has to contemplate caring for its mental patients in a wholesale manner, and I think perhaps they are extremely wise when they finally have decided that they will have a six-thousand-patient hospital rather than six one-thousand-patient hospitals. If the need is there, do not be afraid of a large hospital. It is a question of management and of organization, and a large hospital can do just as good work, and perhaps better work, than some small hospitals.

Mr. Lutgens spoke, and I think it is well for me to refer to it again, of the fact that the old monastic idea of an institution with all its personnel living in the hospital under the immediate control of the superintendent, is something which no longer has to be given consideration. Why should this state spend good, hard American dollars to supply housing for employees who would be much better off and would be much happier if they could live elsewhere independently?

That will be particularly true, Mr. Lutgens, if you ever embark on an eight-hour day, because employees, if they are living on the grounds, necessarily have to play about eight hours of the time, and nobody wants idle employees kicking around.

You need in an institution a certain reservoir of employees to be called upon if there is a great emergency. When you have that, you have all that you need, and the more you get away from the institution, the better off the institution is. The employees, in these days of easy transportation, come in from their homes, perform their services in the hospital and go home again.

Many people will be interested, and I particularly shall be glad to know how these two mental hygiene institutions work out in practice. I think it is an excellent idea. We in New York have had one, but only one. It is a splendid facility, but we have been deterred by the cost. We really need a half dozen. I am glad to see that you are starting with two and I hope they will demonstrate their usefulness to you so convincingly that you will establish others.

I think the Section is greatly indebted to Mr. Lutgens for his excellent presentation of a difficult state problem and the mental institutions are to be congratulated that as the Director of the State Institutions, they have

Mr. Lutgens. He evidently has the welfare of the mental patients completely at heart.

DR. JOSEPH E. BARRETT (Lansing, Mich.).—Mr. Lutgens' paper has presented to us a history of the development and care of the mentally ill in California which is very little different from the history in other states, which brings to us at the present time the picture of great overcrowding which prevails in practically all the institutions throughout the United States and Canada. It does, however, present to us very vividly the place that mental hospitals and the care of the mentally ill have taken in the minds of the public in the past, and makes us realize that the mental hospitals throughout the country have been forced to take what is left after all the other pressure groups have gotten their share out of the state budget.

I am fully convinced that it is high time that those who are interested in mental hygiene and the care and treatment of the mentally ill begin to formulate some pressure groups themselves, so that these conditions are not permitted to continue. I believe that when these matters are brought forcefully to the attention of the legislators we shall get results in relieving this situation. My contacts with members of state governments have led me to know that they are all human and that they want to do these things when they are presented to them in the proper way. I believe that Mr. Lutgens and his organization are beginning to do that in California, and that this whole problem of overcrowding in the institutions will be effectively dealt with.

Mr. Lutgens has spoken of raising the standards of personnel through civil service and higher educational backgrounds. This is very commendable; for the care and treatment of the mentally ill demands the services of the most intelligent groups of the various communities.

There is one thing that I should like to call attention to however, and it has a bearing upon the low per capita cost in California as compared with New York State. As Dr. Parsons has presented that, his ratio of personnel to patients is low; the ratio of physicians and of nurses to patients is low. The total cost of personnel throughout the California hospitals is less, considerably, than 50 per cent of the cost of operation. In those jurisdictions where we think we are doing a good job, the personnel cost throughout the institutions is in the neighborhood of 50 per cent, or better, of the total cost of operation.

One factor that might tend to lower the per capita cost in California as compared with New York, Massachusetts or Pennsylvania, is the climate, which reduces materially the expenditure for heating.

However, I would like to leave with you this idea that, in my opinion at least, the ratio of personnel to patients should be increased. There is every indication for a considerable increase in the number of physicians, nurses and attendants in the institutions. I would appreciate hearing something from Mr. Lutgens in regard to the number of graduate nurses and the ratio of graduate nurses to the total institutional population.

The improvement of salary schedules is to be commended, and should lead to the securing of more highly qualified personnel.

The question of housing personnel on hospital grounds or permitting them to live in the communities is, in my opinion, still an open one. There are certain institutions so located that the housing facilities in the community are inadequate and accommodation at the institution is desirable. In other locations with better community housing facilities a more liberal attitude might be taken. As a matter of general policy it is something which can be decided in each state. Personally, I am of the opinion that from the standpoint of cost over a long time, it is cheaper to house the employees at the institution. I have talked with Mr. Lutgens and he has indicated to me that as a result of his investigations he is convinced that to permit employees to live out can be shown to be a definite saving to the commonwealth, and I hope I may be able to secure the data on which he arrives at that conclusion.

The total annual increase in population throughout the hospital system, of approximately 800 patients seems rather high, and I think has a definite bearing upon the ratio of personnel to patients. There is no question that the front doors of the institutions in California are wide open. This large annual increase in the total population strongly indicates to me that the outlet is not so widely open. That is probably the result of inadequate personnel both in the medical and nursing staffs, and also in social service supervision.

We have all known of California's system of deporting patients to other states, whereby patients accumulate for a period of a year or 16 months and are then deported in herds. Personally I prefer the more individual effort of deporting patients. Whether it is a saving to California or not, is something to be considered; but patients who remain in the institutions for a year, or 16 or 18 months, run up the cost of maintenance while waiting to have a trainload of 200, and that cost is probably as great as if they were deported individually, with the advantages of adequate supervision during the trip.

Mr. Lutgens presents to us a highly qualified layman's view of mental hospitals, and I believe that he has good understanding of the problems of caring for and treating the mentally ill in California. I think, personally, that he would do well to associate with him in his central department medical advice and supervision in an effort to standardize and bring to the highest level throughout his hospital system the medical care and treatment of patients.

In that connection I should like to make just a comment on the function of a state hospital. I don't know how the two medical centers mentioned are going to work out. Personally, I have the feeling that there is a great likelihood of these two medical centers detracting a great deal from the prestige of the individual institutions. In my opinon a mental hospital should be so equipped as to receive, care for and treat properly all the patients in the district which it serves. In addition each hospital should be responsible for the mental health of the district which it serves. It should be the source

of psychiatric information to the general practitioners and to the laity and should carry on at all times educational programs throughout its district.

I think each hospital should be more or less self-contained so far as the service to its district is concerned, and with these two highly specialized centers receiving all patients committed from a certain portion of the state, it will be necessary to guard against the idea on the part of the population of that district that when a patient leaves this particular medical center and goes to a state hospital, his case becomes more or less hopeless and the institutions come to be looked upon as custodial places, regardless of the fact that they have some facilities for care and treatment.

I fear that there is going to be a tendency to concentrate all that is new, all that is progressive, in the field of treatment, in these two centers, and to let the individual institutions suffer from lack of equipment, lack of personnel, lack of adequate treatment and care.

DR. PHILIP SMITH (New York, N. Y.).—You have heard our former Commissioner speak about the work carried on in New York State, and I shall be interested in Mr. Lutgens' remarks in regard to influx of population in California, which tends to increase the population of the state institutions. In New York State we have a constant influx from foreign countries because everything gravitates to New York.

You have heard a great deal about per capita cost: In New York State institutions this varies, but it is never lower than $2 a day and in some institutions it reaches over $3 a day. That, of course, means that the New York people, for any non-resident who remains there, or for any patient who is a foreigner, have a potential liability for keeping those patients in our institutions sometimes 20 or 30 years. In former times, when deportation was not carried out, we found that some of these patients remained almost half a century. You can see, therefore, that to the taxpayers of New York State such a patient, at $2 a day, is a potential liability of $30,000 to $40,000. In the New York State institutions we have on our books about 90,000 patients all suffering from some form of mental disorder. I believe the annual appropriation from the legislature amounts to $33,000,000 for the support of these patients, and that is a tremendous burden upon the state.

We have had a great deal of overcrowding and this developed insidiously and finally led to a situation in which we have oftentimes had as many as three or four hundred patients in some institutions sleeping on floors or in places which were absolutely inadequate for them; therefore, a building program was started and is still in operation. The legislature appropriated on one occasion a bond issue of $50,000,000 which enabled us to erect several very nice, large, new, modern institutions; and again we had another bond issue of $100,000,000, a portion of which gravitated to the state institutions. Overcrowding has been somewhat relieved. Some of the institutions have none and in others it amounts to only a little. Patients no longer have to lie upon the floor, nor do we have to utilize space which is not suitable for the proper care of mental patients. Some of our institutions are old, and we have had to make a sort of tentative provision in what

we consider the amount of space necessary for each patient. We have day rooms, recreation rooms and sleeping quarters. The amount of space which is utilized for these purposes varies in each of these older institutions.

The personnel I think should be increased; during the past year or two we have had the eight-hour day of through shifts adopted. The average is about one employee to eight patients. These employees must be distributed and it means that in some wards we have as many as 80 patients under the control and supervision of one attendant, while in the more acute reception services we oftentimes have as many as two attendants to three or four patients. Occasionally a patient may have a special attendant.

Most of our employees live in the institution. Dr. Parsons has given you a very good picture of our state institutions. He states that he feels that it is no longer necessary to have everything grouped in one place, and I myself feel that it probably would be better if we could give a great deal more leeway in permitting our employees to live outside on the eight-hour basis. I have been in some of the institutions in Europe, and that plan works very efficiently.

We have the eight-hour day for private nurses in New York at $5 for the eight-hour period, and that means if you get a private nurse to take care of a patient, you pay $15 a day.

The big problem is overcrowding and its solution lies, I think, in the direction of prevention. We have instituted in New York State a department which is not in full operation. Through our outpatient clinics, also by making the hospital the center from which emanate all psychiatric activities, by having our doctors speak to organizations throughout the state, we are endeavoring to instruct the public as to what may be done toward preventing the occurrence of insanity, what are the causal factors and how they may be reduced or eliminated.

In my present work as inspector of the Department of Mental Hygiene, I come in contact with many of the other states in regard to non-residents, and a great problem always facing us is the return of non-residents who properly do not belong in the state. I am glad to say that we have a very good working agreement with California, by which certain conditions are set forth which each state maintains, and to those who are here from other states, I should like to say that I hope you will spread this doctrine, namely that we would like to execute more of these mutual agreements with the different states. I think many of the present difficulties will be obviated and better cooperation will be obtained by each accepting the conditions and duties which are set forth in these agreements.

Dr. Samuel W. Hamilton (New York, N. Y.).—There are two points in this admirable presentation of the state of affairs in the California organization that I should like to mention, both with commendation. These are two points that have to do only with building.

The first is that they are building one-storey structures. I am glad that the spirit of Charles S. Little has not passed on with the end of his life. I do not believe that California is the only climate in which one-storey

structures are economical, besides being vastly better for the patients. It is a pleasure to know that California is doing this.

The second point is that California is not content with only large institutions and is now planning two institutions of not over four hundred bed capacity. The advantages of hugeness are often overrated.

DR. FREDERICK W. PARSONS (New York, N. Y.).—I do not want the people in other states to think that Dr. Smith and I are in disagreement. I said the per capita cost was $1 a day, and that is personal service and maintenance cost. Dr. Smith says $2 a day; that means, in addition, interest on the investment, depreciation and extraordinary repairs. We each understand each other, but I want you to know the distinction.

MR. HARRY LUTGENS (Closing).—I don't want to prolong this discussion unnecessarily, but there are a few questions I feel I should answer to Dr. Parsons and Dr. Barrett.

Dr. Parsons brought up the question of per capita cost, answered in part by Dr. Barrett for me, and I appreciate his answer very much. We have discussed this matter at some length. The real reason for the difference in cost, I think, is the fact that we have a different climate in California, most of our living being outdoors during the daytime the year around. We have noticed, moreover, a decided decline in cost since we have initiated the food control system, in cost of individual institutions in the purchase of food.

The reason for the six-thousand-patient institution in Southern California at Camarillo is that there are only two other mental institutions in the southern portion of California, while there are four in the northern portion and the southern portion has a considerably larger population than the other part of the state.

Dr. Barrett suggests that we should lobby. I think that is the one reason we have been so successful in getting appropriations for our mental hospitals in the past three and a half years. We started with the bond issue and publicized that from one end of the state to the other and the people became acquainted with our problem.

I pointed out that one of the defects in our state mental hospital situation in California was that the people knew nothing about it. You are professional men and you have your standards and when you do something you should tell the public about it, and in that way you would awaken interest in the hospital services and make it easier to expand it.

Dr. Barrett brought out the personnel problem in the State of California, that is, the ratio of personnel. I don't know whether we are under-staffed or not. There are a lot of things I have to learn, but I do know this:

we have one physician to each 300 patients, and one attendant to each 12 patients, and our entire personnel averages one to six and a half patients. Whether that is too low or not, I don't know. I think we need more doctors, and I am strongly in favor of increasing the number, and I favor also a larger number of psychiatric social service workers.

I am deeply appreciative of the remarks of Dr. Parsons and Dr. Barrett, and wish to thank you for this opportunity of appearing before you.

ESTRIN EXCRETION IN PSYCHOTIC PATIENTS.*

By JOHN LANSBURY, M. D., and JOSEPH HUGHES, M. D.,
Philadelphia, Pa.

There exists at the present time considerable speculation as to the relationship of the glands of internal secretion to the psychoses. The phenomenon of amenorrhea, which is commonly encountered during the courses of these states, naturally points to the possibility of some disturbance in the production or secretion of the ovarian hormone (estrin).

Bio-assay of estrin in cases of schizophrenia has been performed by several workers as noted here. Saethre [1] in 1936 was unable to demonstrate estrin in the urine of a series of women suffering from dementia præcox. Georgi and Fells [2] in the same year made assays of the estrin output during complete menstrual cycles in the case of three female schizophrenics. The total monthly yield was respectively zero, 23 and 100 mouse units (m. u.) of estrin, whereas, by their same method (benzol extraction) a normal control yielded about 850 m. u. of estrin (as recalculated from their chart). In 1935 Kafka [3] made parallel studies of estrin in the blood and urine in the case of one female schizophrenic patient. He was unable to find estrin in the urine, but found an excess of estrin in the blood. Sears *et al.*[4] in 1937 assayed the blood-estrin a few days before the menstrual period in the case of 186 female schizophrenic patients and 50 normal women (as controls). Their findings are striking, in that estrin was demonstrable in 92 per cent of the control patients, whereas it was only detected in 30.65 per cent of psychotic patients.

With the exception of the one case of Kafka, all these studies, both in blood and urine, indicate either a complete absence or a great diminution of estrin in female patients suffering from schizophrenia. In some of the cases studied the menstrual periods were

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regular, and in others amenorrhea existed at the time of the assay or prior to it.

The work reported below represents a study of the estrin excretion over a full intermenstrual period in the case of seven female psychotic patients, four of whom had menstrual irregularities concurrent with their mental illness. Of the group of patients studied, five had schizophrenia, one was of the manic-depressive, depressed type, and one had a psychosis which was unclassified. Their ages varied from 18 to 36 years.

Method.—Assays of the estrin content of the total output of urine between successive menstrual periods were made in all instances. In four patients, assays were performed at intervals of three or more days on the total intervening urinary output, so that an estrin-excretion curve was obtained. In three patients, all the urine was saved during the intermenstrual period, and assays were performed on aliquots of this, and thus the total estrin output calculated for the period.

The method used was a modification of that employed by Frank and Goldberger:[5] 500 cc. of urine, pH 3.5 extracted by mechanical shaking with two equal (500 cc.) volumes of chloroform during a period of one hour each, at room temperature. Residue is obtained by water-bath evaporation of chloroform to dryness and taken up in olive or sesame oil: 0.4 cc. of this injected twice daily for five injections into spayed adult female mice. Vaginal smears are examined thrice daily and pro or full estrus taken as end-point. When a positive smear is obtained, the oil is diluted, and the assay is calculated on the basis of the highest dilution giving a positive smear. Using this technique, our usual daily yield in non-psychotic patients has been about 15 to 25 mouse units. It should be noted that the estrin so measured represents only the "free" or "unbound" fraction.[6]

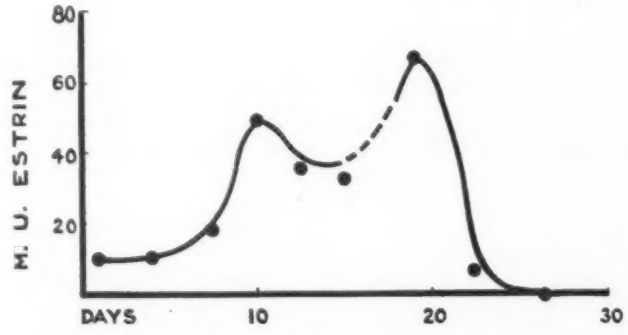
Results.—Data on the amount of estrin excreted by five patients over an interval of days is given in Table 1. The total estrin referred to in this table averaged 23 m. u. per day, and is therefore essentially normal.

Estrin excretion curves determined on four of these patients are presented in the following graphs along with abstracts of the case histories. All curves but one show a characteristically normal peak

between the 18th and 22nd day.[7] Case 1 also shows a secondary peak at the presumed time of ovulation. This is frequently, but not always, found in normal curves.[8] In Case 2 the peak occurs 16 days before the onset of the next menstrual period. This is of interest since the patient had been amenorrheic, and the subsequent period seems to have occurred "out of step" with the hormone-

TABLE 1.

Case.	Total days.	Sum of assays, m. u.	Yield per day, m. u.	Assay of pooled 10 per cent portions (×10),
1	24	586	24.4	....
2	24	570	23.7	774
3	31	591	19.0	712
4	31	796	25.6	1247
5	29	...	...	866



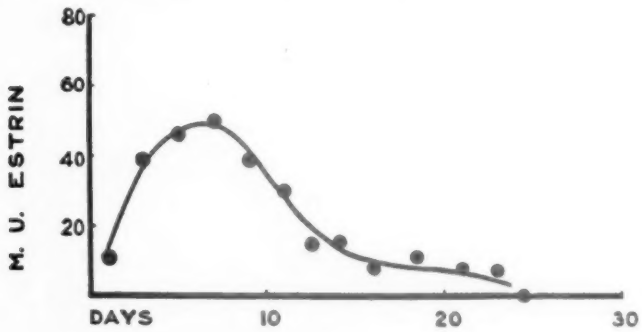
CASE 1.

Patient B. S. age 25. Diagnosis: schizophrenia. Duration six months. Missed one menstrual period three months before test. Weight normal, physical examination, including pelvic examination, X-ray of sella, basal metabolism, complete blood count, and urinalysis—negative.

curve. Cases 3 and 4 show the expected rise in estrin excretion in the latter half of the menstrual cycle.

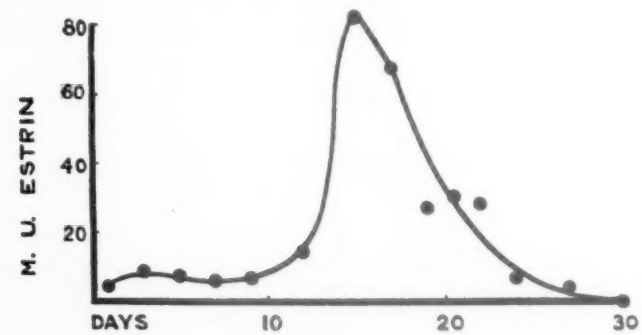
To confirm the accuracy of the above excretion curves, 10 per cent portions of each specimen as it was received were set aside and pooled. Samples from these combined fractions were assayed in the same manner. The results given in Table 1 show a fairly constantly greater amount. This may be due to a gradual liberation of bound estrin in a strongly acid medium over a period of weeks.

In Table 2 are summarized the total estrin outputs per month in the case of two female psychotic patients. The urine for these assays was collected daily, and in each case kept in a common vessel



CASE 2.

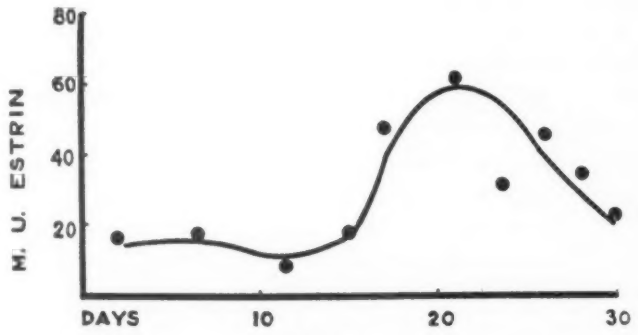
Patient M. W. age 28. Diagnosis: manic-depressive-depressed. Duration five months. Two menstrual periods preceding test had been missed. Physical examination showed moderate generalized hirsutism of the lanugo type, and slight secondary anemia. The pelvic examination, basal metabolism, and urinalysis were all normal. X-ray of sella negative.



CASE 3.

Patient C. C. age 36. Diagnosis: psychosis unclassified. Duration 10 months. Menstrual periods regular. Physical examination including pelvic examination, X-ray of sella, complete blood count, urinalysis, basal metabolism was negative.

until the full intermenstrual output was obtained. The specimens were kept on ice, and, by addition of small quantities of concentrated hydrochloric acid, were kept acid to litmus. Four 500 cc. samples were then assayed, and the results averaged.



CASE 4.

Patient H. M. age 21. Diagnosis: schizophrenia. Duration five months. Missed menstrual period immediately following test. Physical examination negative except for a rather small uterus. Urinalysis, complete blood count, basal metabolism, and X-ray of sella were all negative.

CASE 5.

Patient A. C. age 27. Diagnosis: schizophrenia. Duration 10 months. Amenorrheic for 5 months preceding test. Had first period during the test, and two succeeding periods were normal. Physical examination essentially negative. Urinalysis, complete blood count and sella X-ray negative. Basal metabolism + 17 per cent on one occasion. Total intermenstrual estrin excretion 866 m. u. See Table 1.

TABLE 2.

Case.	Total days.	Total estrin, m. u.	Yield per day, m. u.
6	28	2436	87.0
7	28	2504	89.4

CASE 6.

Patient B. J., age 18. Diagnosis: schizophrenia. Duration 19 months. Menstrual periods regular. Physical examination, including pelvic examination, normal. Urine analysis, complete blood count, X-ray of sella all normal. Basal metabolism normal.

CASE 7.

Patient E. K., age 21. Diagnosis: schizophrenia. Duration 5 months. Menstrual periods irregular—intervals of 16 to 32 days, but no periods of amenorrhea. Physical examination, including pelvic examination negative. Urine analysis, complete blood count and sella X-ray negative. Basal metabolism normal.

It will be noted that the yield in this series is considerably higher than in the first series. This is to be expected since our previous technique was altered in three respects, viz.: (a) The hormone was treated by adding 75 cc. of conc. HCl to 500 cc. portions of urine and boiling in a reflux condenser for two hours before extracting with chloroform, according to the method of Smith & Smith,[6] thus freeing the "bound" estrin which is otherwise inactive. (b) Individual injections were smaller (0.05 cc. instead of 0.40 cc. as before) thus permitting a greater absorptive surface of oil per unit of contained hormone. (c) The site of injection was sealed with collodion, thus reducing leakage to a minimum. In reading the vaginal smears, full cornification was taken as the end-point and, in most cases, it was accepted as the end-point only when present in four out of six mice.

In considering the results shown in Table 2, one may note that the monthly estrin output is fairly constant. The recorded amounts are in the same range as those reported by Taylor [9] using essentially the same technique in assaying the urinary estrin of non-psychotic patients. From the viewpoint of total estrin output, these larger quantities are perhaps more accurate than are the smaller quantities obtained by the first method, but it must be remembered that the greater yields are probably due to the activation of bound estrin by hydrolysis,[6] and that the physiological significance of free and bound estrin excretion in the urine is not as yet fully understood.

In the case of those assays listed in Tables 1 and 2, it is noteworthy that in all cases but Nos. 3, 6, and 7, periods of amenorrhea occurred.

Summary and Conclusions.

Previous assays of estrin in the urine of female schizophrenics have all indicated either a diminution or a complete absence of the ovarian hormone.

Assays of the female sex hormone (estrin) in the urine of seven psychotic patients are reported here. Four of these patients have been amenorrheic at varying periods. The estrin excretion curves, as well as the total monthly estrin excretion, both free and bound, fall within normal limits in all the cases studied.

The authors acknowledge with appreciation the technical assistance of Miss Winifred Strowhecker and Miss Barbara Smith as well as the cooperation of the nursing staff of the Pennsylvania Hospital in collecting the specimens for analysis.

BIBLIOGRAPHY.

1. Saethre, H.: Klin. Wchnschr., 12: 1409, 1933.
2. Georgi, F., and Fels, E.: Ztschr. f. d. ges. Neur. u. Psych., 147-148: 746, 1933.
3. Kafka, V.: Wien med. Wchnschr., 85: 30, 1935.
4. Sears, H. A., Morter, R. A., Simonsen, M., and Williams, C.: Am. J. Psychiat., 93: 1293, 1937.
5. Frank, R. T., and Goldberger, M. A.: J. Am. Med. Assn., 94: 1197, 1930.
6. Smith, G. V., and Smith, O. W.: Am. J. Physiol., 112: 340, 1935.
7. Frank, R. T., Goldberger, M. A., and Spielman, Frank: J. Am. Med. Assn., 103: 393, 1934.
8. Smith G. V., Smith, O. W., and Pincus, G.: Am. J. Physiol., 121: 98, 1938.
9. Taylor, H. C., Jr.: Surg. Gynec. and Obst., 62: 562, 1936.

METABOLIC DEFICIENCY AS A POSSIBLE FACTOR IN NEUROPSYCHIATRIC STATES.*

By ERWIN WEXBERG, M. D., New Orleans.

Since 1930, when Wechsler [1, 2] first promulgated the theory that many if not all cases of alcoholic polyneuritis might be due to vitamin deficiency, there has been an increasing interest in this possible solution of an important problem. The rather frequent association of neurologic and psychiatric symptomatology with chronic alcoholism is well known, as is a similar symptomatology in other cases of dietary deficiency in which, by presumption at least, avitaminosis is a factor.

In *The Southern Medical Journal* for March, 1937, we [3] reported a group of 47 cases in which a neurologic and psychiatric symptomatology was associated with a proven or highly probable metabolic pathogenesis. In this communication we pointed out that our conclusions were frankly not well founded in certain cases in which we had only the case history and the therapeutic test to rely upon. At the same time we warned against the danger of undue simplification, that is, the reference of all nervous symptoms in all such cases to nutritional deficiency. An all-inclusive theory of pathogenesis, we pointed out, would fail to account for special characteristics differentiating diseases in special groups from each other, as well as from diseases of other groups.

At the present time our theory is purely speculative. Until some specific chemical or biological test for avitaminosis is available, the classification we have suggested cannot be put on a scientific basis. The single test which has been proposed for avitaminosis B1, by Kimura,[4, 5] has not been specific in our hands, though the number of our cases is admittedly very small.

The 47 cases reported in our first communication have now increased to 62. All of them represent acute and subacute psychoses

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of the organic reaction type, in some instances combined with either peripheral or spinal neurological syndromes such as polyneuritis, posterolateral sclerosis, transverse myelitis or spastic spinal paralysis.

TABLE 1.

NEUROPSYCHIATRIC SYNDROMES IN RELATION TO THE CAUSATIVE METABOLIC FACTORS IN 62 CASES.

Diagnosis.	Alcohol.	Gastro-intestinal disturbances.	Starvation.	Vitamin deficiency.	Puerperal state.	Infection.	Unknown.	Total.
Acute psychosis (amentia)	..	1	1	..	..	1	2	5
Acute psychosis with polyneuritis	..	1	..	..	..	..	1	2
Acute psychosis with pellagra	..	..	..	1	..	..	..	1
Alcoholic psychosis	7	..	..	..	..	..	..	7
Alcoholic psychosis with polyneuritis	5	..	..	..	..	..	..	5
Alcoholic psychosis with polyneuritis and pellagra	2	..	..	..	..	..	..	2
Alcoholic psychosis with posterolateral sclerosis	1	..	..	..	..	..	..	1
Korsakoff syndrome with transverse myelitis	1	..	..	..	..	..	..	1
Korsakoff syndrome, posterolateral sclerosis and pellagra	..	..	1	..	..	..	..	1
Polyneuritis	13	2	1	2	1	2	4	25
Polyneuritis and pellagra	1	..	..	..	..	..	..	1
Transverse myelitis	2	1	..	..	..	..	2	5
Posterolateral sclerosis	..	..	1	..	..	..	3	4
Spastic spinal paralysis	1	..	..	..	..	..	..	1
Posterolateral sclerosis and anterior horn involvement	..	1	..	..	..	..	..	1
Total	33	6	4	3	1	3	12	62

The various conditions included in this study are set forth in Table 1, in relation to the demonstrable or presumable etiologic factors. In most cases the psychiatric syndromes observed were those of acute, so-called toxic psychosis, amentia or delirium. There are 13 cases of mental disease without demonstrable neurologic findings, 12 cases of psychoses with neurological findings (spinal or peripheral nervous disease), and 27 neurologic condi-

tions without psychoses. There are also eight cases of posterolateral sclerosis included in our material, the pathogenic aspect of which should be particularly commented upon. In two of the eight cases in this group alcoholic psychoses were present, and we are inclined to wonder whether the association is merely a coincidence. This particular entity is no longer considered due to pernicious anemia, of which there was no evidence in any of our cases, but rather is regarded as a coordinated syndrome due to the same deficiency factor which may produce pernicious anemia, secondary hypochromic anemia and achlorhydria. The deficiency in all of these conditions is believed to be an unknown liver factor, or an unknown gastric mucosa factor or both. If the metabolic pathogenesis of this spinal syndrome be granted, alcoholism may be accepted as an occasional indirect causative factor, through alcoholic involvement of the liver or the stomach or both. Such a line of reasoning could be applied to explain the combination of posterolateral sclerosis with alcoholic psychosis.

Several cases of delirium tremens are included in this analysis, not because we question in any way the specific alcoholic etiology of this disease but because a combined toxic-metabolic pathogenesis seems reasonable in some cases, at least. The same argument can be applied to Korsakoff's syndrome associated with alcoholism, though it is freely granted that here the alcoholic etiology is not quite so specific. In one such case in this group transverse myelitis was also present. The combination might be considered merely a coincidence except for the fact that we have seen two other cases of transverse myelitis in which alcoholism was the only demonstrable etiologic factor. The repeated association seems to warrant the suggestion that perhaps in some cases of transverse myelitis of unknown etiology, particularly when alcoholism is associated, avitaminosis may be thought of as an indirect causative factor.

From the standpoint of etiology, 33 of the 62 cases were definitely alcoholic in origin. In 12 cases no definite cause could be determined. The other 17 represented such varying etiologic factors as gastrointestinal disturbance (6), starvation (4), frank avitaminosis (3), nonspecific infection (3), and the puerperal state (1).

Thirty-nine patients have been followed for sufficiently long periods of time to permit some estimate of the permanent value of the treatment. Of the 32 treated with a high-vitamin diet, on the

TABLE 2.

RESULTS OF TREATMENT IN 42 FOLLOWED-UP NEUROPSYCHIATRIC CASES.

Diagnosis.	Vitamin diet. Improved.	Vitamin diet. Unimproved.	Regular diet. Improved.	Regular diet. Unimproved.	Died.*	Total.
Acute psychosis (amentia)	1	..	1	..	..	2
Acute psychosis with polyneuritis	1	..	..	..	1	2
Acute psychosis with pellagra	..	..	..	..	1	1
Alcoholic psychosis	4	..	1	..	..	5
Alcoholic psychosis with polyneuritis	2	..	..	..	..	2
Alcoholic psychosis with polyneuritis and pellagra	2	..	..	..	..	2
Alcoholic psychosis with transverse myelitis	..	..	..	..	1	1
Alcoholic polyneuritis	6	..	1	3	..	10
Polyneuritis	9	..	..	..	..	9
Polyneuritis with pellagra	1	..	..	..	..	1
Transverse myelitis	2	1	..	..	..	3
Posterolateral sclerosis	2	..	1	..	..	3
Posterolateral sclerosis and anterior horn involvement	1	..	..	..	..	1
Total	31	1	4	3	3	42

* All deaths were due to intercurrent disease.

TABLE 3.

RESULTS OF TREATMENT IN 42 FOLLOWED-UP NEUROPSYCHIATRIC CASES IN RELATION TO THE ETIOLOGICAL FACTORS.

Cause.	Vitamin diet. Improved.	Vitamin diet. Unimproved.	Regular diet. Improved.	Regular diet. Unimproved.	Died.	Total.
Alcohol	15	..	2	3	1	21
Gastro-intestinal disease	5	..	..	..	..	5
Starvation	1	..	..	..	..	1
Vitamin deficiency	2	..	..	..	1	3
Puerperal state	1	..	..	..	..	1
Infection	3	..	..	..	..	3
Unknown	4	1	2	..	1	8
Total	31	1	4	3	3	42

theory that avitaminosis was a specific factor in their illness, only one, a man with polyneuritis of unknown origin, failed to improve. Of the seven patients who received an ordinary diet four improved, and three, all of whom had alcoholic polyneuritis, did not. This disease has an excellent prognosis with a high-vitamin diet, and the improvement noted in the four patients on ordinary diet may perhaps be attributed to the fact that even the unsupplemented hospital diet is probably richer in vitamins than the average diet of the class of patients treated at Charity Hospital in New Orleans. Three patients, not included in the 39 cases just mentioned, died of intercurrent disease.

The mental conditions observed in these patients were widely varied. They included states of confusion or delirium, with consciousness more or less clouded, visual or auditory hallucinations and delusions, as well as states of anxiety, perplexity and severe depression. At the height of confusion hypomanic conditions frequently occurred, with increased psychomotor activity, restlessness and flight of ideas. Some cases of catatonia-like stupor were also observed. One occurred in an 11-year-old boy, in whom a psychosis with catatonic features developed after typhoid fever. The psychosis lasted for six weeks but subsided, almost abruptly, when high-vitamin dietary therapy was instituted. Another typical case occurred in an 18-year-old male, who was admitted in a stuporous condition which suggested schizophrenia. Two facts, however, were definitely in favor of another interpretation. The patient was obviously undernourished, and a history was secured of an inadequate diet for some months before the present attack. Neurologic examination revealed an absence of both patellar and achilles tendon reflexes, findings which, with an otherwise negative neurologic examination, could be accounted for only by assuming the presence of a polyneuritis. Recovery was prompt after the institution of a high-vitamin diet.

It would be a mistake, merely on the basis of our personal observations and of other observations in the literature which we noted in our first communication, to overstress the importance of vitamin deficiency in neuropsychiatric states. The mere fact that high-vitamin therapy, particularly the use of vitamin B preparations, has established its curative value, does not necessarily prove the cause and effect relationship. As a matter of fact, in our own

group of cases it was possible to elicit a frank history of vitamin deficiency in only three cases. It might be fair to assume a similar deficiency in some of the other cases, but it is scarcely reasonable to assume it for all of them. For this reason we have tried to replace the specific conception of avitaminosis by the more general conception of "neurometabolic crisis."

By the term neurometabolic crisis we understand three things: (1) that the processes involved are essentially neurologic, with various localizations; (2) that metabolic derangement is one of the pathogenic factors, and perhaps the most important; (3) that the process has, primarily, the character of a crisis, that is, of an acute or subacute disturbance which may end in recovery or in an irreversible degenerative process. The pathologic processes may consist of cellular starvation or asphyxia of nervous elements, and may be produced by: (a) starvation, (b) disturbances of gastrointestinal absorption, (c) absolute or relative avitaminosis, often following alcoholism, (d) pregnancy and the puerperal state, (e) various metabolic diseases such as diabetes, (f) various endocrine diseases such as hyperthyroidism, (g) cachexia associated with such diseases as phthisis pulmonis or carcinomatosis.

For some reason which is not yet clear the neurometabolic conditions which we are discussing are usually reversible when they are located in the brain, where they are manifested as psychoses, or in the peripheral nerves, where they are manifested as polyneuritides. When they are located in the spinal cord, on the other hand, and are manifested as posterolateral sclerosis, transverse myelitis, and spastic spinal paralysis, they tend to become irreversible. In one case of acute psychosis and polyneuritis a very careful necropsy was performed by Dr. Gabriel Steiner, who reported that examination of the brain was entirely negative.

The underlying causes of neurometabolic crisis which we have listed are for the most part obvious and need no special explanation. Under disturbances of gastrointestinal absorption, for instance, may be included other diseases which produce somewhat the same effects, such as dysentery, typhoid fever and nonspecific gastroenteritis. The neurologic symptoms of post-dysenteric polyneuritis, with or without amentia, and those of beri beri are so similar that we do not believe the presence of a specific infectious agent in dysentery justifies an etiologic distinction between them.

Typhus, influenza, pneumonia and other infectious diseases which occasionally produce similar neuropsychiatric syndromes may act directly, by toxic involvement of the nervous system, or indirectly, as the result of pyrexia and hyperpyrexia. Under such circumstances the increased metabolic rate is responsible for an increased vitamin requirement; if the diet contains merely the average vitamin content, a relative avitaminosis is promptly produced.

Pregnancy, again, acts as a causative factor through the production of a relative avitaminosis by reason of the additional vitamin requirement of the fetus. In the puerperium the metabolic stress of pregnancy, the fatigue of labor and postpartal endocrine readjustment, may all be factors. An increased metabolic rate, with an increased and unmet vitamin requirement, may play some part in the psychotic episodes of thyrotoxicosis. Psychoses with or without neurological signs which occur in cachectic states may also be due to a metabolic deficiency of the nervous system, as part of the general critical condition of body tissues. Gastrointestinal carcinoma is, for obvious reasons, more often associated with neurometabolic crises than malignancy of other locations.

Such functional psychoses as schizophrenia and manic-depressive psychosis are sometimes associated with quasi-organic reactions, and it is not unreasonable to suppose that a neuro-metabolic crisis may be one of the contributing factors. The chain of events may be pictured as a system of overlapping or vicious circles. First comes the premorbid personality, which includes the unknown entity of the constitution. Upon it certain psychologic factors may or may not intervene. In many cases their influence is clear. They account in part for the emergence of a state of vegetative-emotional tension which may be manifested as anxiety and which, even unaided, constitutes a powerful pathologic-physiologic force, with tremendous repercussions through the organism. Cannon has described the mechanism caused by adrenemia as an emergency reaction, essentially identical with the mechanism and reactions characterizing the emotion of fear. There is a critical metabolic upheaval which is indicated by an increased blood sugar content and similar definite changes. Under normal conditions the equilibrium is reestablished more or less promptly. If other factors intervene, however, it may continue unbalanced. Such factors in-

clude various gastro-intestinal disturbances, starvation, perhaps induced by the patient's own refusal to take food, alcoholism or a diet low in vitamins. These factors, which are widely different in most respects, are similar in one, that they may interfere with the self-regulatory mechanisms which normally reestablish metabolic equilibrium and may thus ultimately produce an additional imbalance. The disturbance due to the emergency reaction then becomes a vicious circle which is maintained by psychological as well as by physical factors. Ultimately there is produced the neurometabolic crisis.

In the same way, the tremendous psychic shock of psychotic experiences, such as hallucinations or delusions, involves an additional psychologic imbalance, with constantly increasing emotional tension. This is another source of metabolic disturbance, and another reason for failure of reestablishment of equilibrium. It is presumably the different constitutional background which accounts for the different outcome in such cases: one patient will develop chronic progressive disease, another periodic insanity, and a third delirium followed by complete recovery.

Shock therapy of some sort will sometimes break the vicious circle we have described, or end an acute or subacute psychotic episode, and thereby give the organism an opportunity to reestablish normal equilibrium. It cannot, however, change the constitutional background of a schizophrenic. Recurrence of the psychosis may follow another emotional and metabolic unbalance, and this is almost inevitable, sooner or later, unless the patient happens to live under especially favorable conditions. Psychologic as well as economic factors are concerned, for experience has shown that the prognosis of schizophrenic patients in a state of remission depends largely upon the environment to which they return after their discharge.

In certain cases the first attack of schizophrenia cannot be distinguished clinically from a confusional state of toxic origin. The only real distinction is that some acute psychoses in young individuals clear up and leave no residue, whereas others lead to chronic and incurable illness. The outcome is probably determined by some constitutional factor. The psychotic process itself, however, may easily be the same in both instances. One is justified, therefore, in employing the same treatment tentatively in all these

cases when they are first seen, whether one is dealing with an amentia associated with alcoholic hallucinosis, or with a first attack of paranoid or catatonic schizophrenia. The attempt to reestablish the metabolic equilibrium by a high-vitamin diet and the administration of special vitamin preparations do no harm, do not interfere with other methods of treatment, and may be productive of considerable benefit. It seems reasonable, therefore, to employ it tentatively on all such patients.

In the cases we are reporting vitamin therapy seems to have been an important curative factor. The series is naturally too small to permit of a comparison of this type of treatment with other types, and for a scientific proof of our thesis, we have pointed out, we must await the development of some specific test for avitaminosis. We can fairly say, however, that the so-called toxic psychoses, delirium, amentia, certain instances of Korsakoff's psychosis, the psychoses of pellagra, and related conditions clear up more rapidly with a high-vitamin diet than without it.

SUMMARY.

1. Sixty-two patients were studied in whom organic disease of the central nervous system was probably induced, at least in part, by metabolic deficiency. Mental symptoms were present in 25 of these cases.

2. The deficiency is termed a "neurometabolic crisis." It may be caused by absolute or relative avitaminosis, by starvation of various origins or by endocrine factors.

3. Causation of the mental symptoms in certain cases is outlined, schematically, as a complex system of vicious circles, in which the two factors of anxiety and metabolic imbalance both play a part.

4. In view of our personal good results in such cases, a high-vitamin diet is recommended for tentative trial not only in acute or subacute psychoses characterized by delirium and amentia, but also in acute episodes of the organic-reaction type which occur in the course of schizophrenic and manic-depressive psychosis.

BIBLIOGRAPHY.

1. Wechsler, I. S.: Unrecognized cases of deficiency polyneuritis (avitaminosis?). Med. Jour. and Rec., 131: 441-444, May, 1930.

2. Wechsler, I. S.: Etiology of polyneuritis. Arch. Neur. and Psychiatry, 29: 813-827, April, 1933.
3. Wexberg, Erwin: Diseases of the nervous system caused by nutritional deficiency. South. M. J., 30: 334-342, March, 1937.
4. Kimura, J.: High blood platelet count in lactants as an early sign of avitaminosis B. Tohoku J. Exp. Med., 26: 360-373, October, 1935.
5. Kimura, J.: Intensity of Arakawa's reaction and blood platelet count: change of platelet count on B-vitamin administration. Tohoku J. Exp. Med., 27: 374-389, October, 1935.

DISCUSSION.

DR. SIEGFRIED BLOCK (Brooklyn, N. Y.).—The thought came to me as I was sitting here today reading through the programs of other sessions, that at present there seems to be a movement of using medicines to cure all kinds of psychosis, whether it is with vitamins, a chemical or something of that sort.

A short time ago it was personality study entirely; a short time before that it was for operation and change of environment, and the question of personality was left out.

It is interesting to note how these things go in cycles or patterns.

We are happy that we heard this paper of Dr. Wexberg which emphasizes that we are making more headway in the chemistry and physics, not only of the psychoses, but of the entire human physiology.

Avitaminosis is only one of the many chemical causes of pathology, not only in mental and neurological cases. Of course an analysis of the psyche and general personality traits and development in relation to environment are of most importance. But when the theoretical millennium in medicine comes, and we are convinced of our exactness in the biological phases of our own physics and chemistry, then this whole matter will be very simple. We will be able to give a definite chemical for a known result, and such therapy we now use as general depressants or general stimulants will disappear.

Even the exactness of the individual chemicals which make up any of our ductless glands will be used to produce a very exact endocrine result. We are on the way now to such things with cortin, antuitrin and insulin.

Arteriosclerotic brain conditions are not far removed from essential hypertensions. And those, in turn, are not far removed from certain endocrinological activities. Chronic alchoholics, lead poisoning and arsenic cases are also closely associated with the arteriosclerotics.

Very old so-called functional mental conditions gradually deteriorate and on postmortem show organic brain lesions. *Vice versa*, some organic brains show only what we are prone to call functional disorders. Many of us made substantial improvements in some psychoneurotics by injecting air. Two types of psychoses which I have never seen in print are worth summarizing:

1. Chronic strychnine psychoses: The four cases I have seen came from inhaling strychnine powder in a drug house where strychnine tablets were manufactured. The summary of the symptoms is as follows: (a) The eyes were surrounded by crusty scab with some puffiness of both lids. (b) There

is a sensory polyneuritis. (c) Loss of weight. (d) Tachycardia alternating with bradycardia. (e) Delusions of grotesque character without hallucinations. (f) Anxiety and apprehension. (g) Normal blood pressure. (h) Marked gastro-enteritis with diarrhea, in one case vomiting. (i) All had restlessness and insomnia. (j) Very poor appetite.

2. The other psychoses which I have seen in two cases came from inhaling powdered sugar. Both cases were: (a) Underweight. (b) Had pallor of the skin with secondary anemia. (c) Rapid pulse. (d) Constipation amounting almost to obstipation. (e) Acne. (f) Apprehension. (g) Mild depression. (h) A few catatonic phenomena. (i) No increase of sugar in the urine or blood. (j) Fine tremors of the whole body and tongue. (k) Very low blood pressure; both cases had a diastolic of zero. (l) Tremulous speech. (m) Bronchitis and a dry cough. (n) Very poor appetite.

I saw these psychoses several years ago in a factory where no precautions were taken in making candy out of 4X powdered sugar. There was total recovery without recurrence and without any treatment when the people stopped working.

One more thought comes to me in discussing definite chemicals causing exact pathology. I am reminded when at the Greenpoint Hospital in Brooklyn we had quite a few cases of acute deaths from alcoholic hemorrhagic wet brain.

The patients would be in the hospital from a few hours to a few days, and die from drinking cheap alcohol. The reactions were of such a nature that the orderlies could tell by the condition of the patient where he got his particular "white mule." We all know of an acute strychnine poisoning of definite mental and physical reactions.

Finally, the difference between a deficiency in the chemical supply of the body and the wrong chemical supply to the body is only words. At present, anyway, we must not neglect the personality component. Why did all persons who worked in these factories not become insane? Why did they not all have such an idiosyncrasy? If it is due to a chemical make-up of the personality, we are on the way, but far from our goal as yet.

DR. ERWIN WEXBERG (Closing).—I want to thank Dr. Block for his discussion, and I only want to ask the question whether it might not be possible that those cases of strychnine psychoses might be only secondarily induced by strychnine, and primarily by the gastro-intestinal upheaval which is caused by strychnine poisoning.

The same to some extent might be questioned in the case of the sugar dust psychosis which he mentioned. These cases are extremely rare. I never saw a case of that kind, and they are not to be found in literature, but it might easily be possible that all those damages done to the psyche are due to the metabolic disturbance which is the immediate consequence of the poison, bringing on the psychic disturbance.

MENTAL DISORDERS IN TRIPLETS.

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In the spring of 1930 we undertook to collect material for a study of mental disorders in twins. The task of collecting this material was completed in the early part of 1933, by which time we had gathered records of 1014 pairs of twins with a mental disorder in one or both of the twins in each pair. Since then we have been occupied with the work of analyzing the material and have published our findings, from time to time, by clinical groups.

In going over our material we found ourselves in possession of records of 12 sets of triplets, which had been picked up incidentally; and the object of this communication is to report our study of this part of our material, it having yielded some findings of special interest.

Our studies, up to this point, have already led us to a new and higher appraisal of the importance of cerebral birth trauma in the etiology of certain so-called constitutional mental disorders; namely: infantile cerebral palsies; mental deficiency;[1] epilepsy;[2] behavior difficulties of children, juvenile delinquency and adult criminality; and some deteriorating psychoses of adolescence and early adult life.[3] Also, we have had occasion to point out that neuropsychiatric conditions which are seen as residuals or sequels of cerebral birth trauma are relatively more common among twins than among singly-born subjects.

The higher incidence of these conditions in twins is attributable, of course, to the greater vulnerability of twins, which, in its turn, is due to the much greater frequency of premature birth and underweight condition at birth among twins as compared with singly-born subjects.

Just as the hazards of prematurity and immaturity are greater for twins than for singly-born subjects, so they are still greater for triplets and quadruplets. Accordingly, we may expect, *a priori*, to find, among triplets and quadruplets, relatively high percentages

of stillbirths, and, in surviving subjects, a relatively high incidence of neuropsychiatric residuals and sequels of cerebral birth trauma.

The numbers of stillbirths per 100 live births for single and plural births, as calculated on the basis of statistical data furnished by the U. S. Census (1930), are as follows:[4]

Single births	3.4
Twins	7.9
Triplets	16.8
Quadruplets	25.0

As is well known, triplets may be monozygotic, dizygotic or trizygotic. In our collection, two sets are probably monozygotic, four rather definitely dizygotic and the remaining six trizygotic.

Of the 36 triplets (individuals) represented in our material, 21 are males, 15 females, distributed in the sets as follows:

3 males	2 sets
2 males, 1 female	7 sets
1 male, 2 females	1 set
3 females	2 sets

Among these subjects, three were stillborn or died soon after birth: two males, one female; leaving 33 surviving subjects comprised in our material.

About one-half of the subjects (16 out of 33) are of subnormal intelligence (I. Q. 60 to 89); and, as among both twins and singly-born subjects, the incidence of subnormal intelligence is higher among the males than the females: 13 (68.4 per cent) and three (21.4 per cent), respectively.

In 12 of the subjects (eight males, four females) other neuropsychiatric conditions were noted in addition to the subnormal intelligence, as follows:

Clinical conditions.	Males.	Females.	Total.
Child behavior difficulties	4	2*	6
Juvenile delinquency	..	1	1
Adult criminality	1	..	1
Chronic psychosis	1	..	1
Epilepsy	2	1	3
Total	8	4	12

* One of these in a subject with normal intelligence (I. Q. 95).

There is very little concordance of findings among the triplets of the respective sets. The most significant comparison, as in cases of twins, would be, of course, that of monozygotic with dizygotic pairs. For this purpose the individual triplets in each set are arbitrarily designated as Nos. 1, 2, and 3; and in each set three comparisons are made: 1 with 2; 1 with 3; and 2 with 3. Only one comparison is to be made, of course, in each of the three sets in which one of the triplets was stillborn or died soon after birth. Thus, our material affords an opportunity of a total of 30 such comparisons in the 12 sets of triplets, as follows:

Monozygotic pairs, males	3
Monozygotic pairs, females	3
Same-sex dizygotic pairs, males	8
Same-sex dizygotic pairs, females	2
Opposite-sex (dizygotic) pairs	14

With reference to the existence of subnormal intelligence, these comparisons have yielded the following findings:

Degrees of concordance in intelligence.	6 monozygotic pairs.	10 same-sex dizygotic pairs.	14 opposite-sex (dizygotic) pairs.
Both normal (I. Q. 90 or over)	..	2	5
Both subnormal, in similar degrees	2	2	..
Both subnormal, in dissimilar degrees	..	1	..
One normal, the other subnormal	4	5	9

There is no significant contrast here, in frequency of concordance, as between monozygotic and dizygotic pairs.

With reference to the existence of neuropsychiatric conditions, other than subnormal intelligence, our comparisons have yielded the following findings:

Degrees of concordance with reference to existence of child behavior difficulties, juvenile delinquency, adult criminality, psychotic disease and epilepsy.	6 monozygotic pairs.	10 same-sex dizygotic pairs.	14 opposite-sex (dizygotic) pairs.
Both affected, similarly	1	..	3
Both affected, dissimilarly	..	..	..
Only one affected	3	7	5
Neither of the pair affected	2	3	6

Here, too, there seems to be no significant contrast, in frequency of concordance, as between monozygotic and dizygotic pairs.

Summary.—Twelve sets of triplets, comprising 33 surviving and three stillborn individuals, were incidentally included, along with over a thousand pairs of twins, collected for the purpose of a study of the etiology of mental disorders. The twins and triplets were selected on the basis of at least one in each pair or set having a mental disorder.

Neuropsychiatric conditions were found affecting 17 of the 33 subjects, consisting of subnormal intelligence with or without the following complications: child behavior difficulties, juvenile delinquency, adult criminality, psychotic disease and epilepsy.

Comparisons of the monozygotic with the dizygotic pairs of subjects among the triplets have failed to reveal any significant contrast with respect to frequency of concordance.

The findings yielded by this material, though limited in amount, serve, nevertheless, not only to confirm the conclusions reached by us from our larger-scale studies of twins, but also to further discount the factor of heredity in the etiology of the conditions under consideration.

The neuropsychiatric conditions found were all of types which are often seen as residuals or sequels of cerebral birth trauma.

BIBLIOGRAPHY.

1. Rosanoff, A. J., Handy, L. M., and Plesset, I. R.: The etiology of mental deficiency, with special reference to its occurrence in twins: A chapter in the genetic history of human intelligence. Psychol. Monogr., Vol. 48, No. 4, Whole No. 216, Princeton, N. J., 1937.
2. Rosanoff, A. J., Handy, L. M., and Rosanoff, I. A.: Etiology of epilepsy, with special reference to its occurrence in twins. Arch. Neur. and Psychiat., 31: 1165, 1934.
3. Rosanoff, A. J., Handy, L. M., Plesset, I. R., and Brush, S.: The etiology of so-called schizophrenic psychoses, with special reference to their occurence in twins. Am. J. Psychiat., 91: 247, 1934.
4. Birth, stillbirth, and infant mortality statistics for the birth registration area of the United States, 1930. Bureau of the Census, Washington, 1934.

THE INADEQUACY OF THE EVIDENCE FOR HEREDITARY PREDISPOSITION IN EPILEPSY.*

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Modern authorities are, for the most part, in agreement that epilepsy as such is not hereditarily transmitted. Instead it is usually stated that a special predisposition to epilepsy is inherited. The basis for this hypothesis has three premises. In the first place, epilepsy is said to be so infrequent an occurrence in brain lesions, *e. g.*, head injuries, as to be less readily explained on an acquired basis than by hereditary predisposition. Moreover, there is no constant brain lesion which causes epilepsy. Secondly, the incidence of epilepsy is reported as being greater among the relatives of those afflicted than among the relatives of "normal" controls. Finally, recent statistical studies of epilepsy in twins favor an hereditary predisposition. The evidence for each of these three premises will be considered separately.

1. The Relation of Cerebral Lesions to Epilepsy.—It has frequently been stated that only a small proportion of injuries to the head lead to seizures, presumably in persons with a special susceptibility. Among 25,000 gunshot injuries of the head, convulsions developed subsequently in only 4.5 per cent, according to Sargent.[1] Likewise in other types of extensive brain pathology convulsions occur in only a small proportion.† This evidence must be subjected to much more detailed analysis. Obviously the same argument might be applied to many other neurologic symptoms.

* Read at the ninety-fourth annual meeting of The American Psychiatric Association, San Francisco, Calif., June 6-10, 1938.

† Lennox and Cobb (Epilepsy, 1928, p. 156) say, "There is no constant anatomical lesion in epilepsy, and only a minority of patients with extensive cerebral pathology have fits. We are forced then to postulate some unknown constitutional element. This abnormal 'convulsive capacity' is presumably present in some degree in each patient with epilepsy."

Only a small percentage of patients with head injuries have hemiplegia, apraxia, or astereognosis. Is a genetic predisposition the reason or is it cerebral localization? In regard to hemiplegia and astereognosis the answer is easy—the focal lesions are known. No one thinks of ascribing astereognosis to an hereditary predisposition merely because it occurs infrequently in gunshot wounds of the head. In epilepsy the neural mechanism is unknown. Still there is evidence that the aforementioned reasoning is no more applicable to recurrent convulsions than to hemiplegia, astereognosis or other neurologic symptoms.

Strangely enough, neurologists have been willing to discuss the relation of epilepsy to cerebral trauma on statistics of injury to

TABLE I.

INCIDENCE OF CONVULSIONS IN BRAIN INJURIES.

Author.	No. of cases.	Per cent with convulsions.
Wagstaffe [2]	377(?)	18.7
Frazier and Ingham [3]	200	14.0
Steinthal and Nagel [4]	639	35.5
Stern [5]	...	20.0 to 50.0
Rawling [6]	425	25.0
Schürer-Waldheim [7]	...	25.0
Baumeyer [21]	340	24.0

the head without inquiry as to whether the brain was actually damaged, and if so, in what part. When Wagstaffe [2] analyzed cases with this point in mind, he found that only 1.6 per cent of gunshot wounds without penetration of the dura led to convulsions; whereas with penetration this sequel occurred in 18.7 per cent. Even higher figures are recorded in other similar analyses of war material (see Table I). One would expect the statistics from different sources to vary, dependent among other things on the type of material and the number of years intervening between the injury and the report. Nevertheless, according to such evidence, the incidence of epilepsy in gunshot wounds of the brain is 20 per cent or more, not an insignificant figure. Reference is not made to civil head injuries in industry and from the automobile, because the type of intracranial damage in these accidents is not as readily ascertainable, nor are such large numbers available

from the experience of single investigators as with gunshot wounds. Likewise in the case of brain tumors the occurrence of convulsions is also high, varying from 15 to 39 per cent in different series (see Table II). Spratling [12] and Holt and Howland [13] conclude that from 35 to 40 per cent of patients with infantile cerebral palsies develop epilepsy. Ford [14] gives comparable figures. All these data suggest that cerebral damage of one type or another bears a significant relationship to epilepsy. If one assumes a hereditary predisposition for these cases, it must be present in from 20 to 40 per cent of patients with head injuries and tumors. The improbability of this is augmented by studying the statistics on localized cerebral injuries and tumors.

TABLE II.

INCIDENCE OF CONVULSIONS IN BRAIN TUMORS.

Author.	No. of cases.	Per cent with convulsions.
Dowman and Smith [8]	100	39.0
Sachs and Furlow [9]	724	20.0
Parker [10]	313	21.6
Sargent [1]	270	30.0
Gibbs [11]	1545	15.6
Müller [18]	164	37.2
Davis and Droegemueller [22]	42 *	36.0
List [23]	300 †	53.0

* Brain abscesses.
† All gliomas and all types of epileptic phenomena recorded, *i. e.*, not only convulsions.

Not as much can be said of localization in head injuries, particularly gunshot wounds, as of other more circumscribed lesions. Still there are a few series which show a greater association of convulsions when certain areas of the brain are affected as contrasted to other involvement. In Frazier and Ingham's [3] cases, 22 of the 28 gunshot injuries with subsequent convulsions were in the parietal area. Although Wagstaffe states that localization makes no difference in the incidence of traumatic epilepsy, it is of interest to note that 24 of his 33 cases are listed as being due to lesions in the frontal, frontoparietal and parietal areas.

The evidence from brain tumors is more clear-cut and convincing than from injuries. Perusal of Table III will show that in tumors of the frontal, temporal or parietal lobes, there are convulsions in from 30 to 80 per cent of the cases in different series.

In this connection it is of interest that in Parker's cases, designated as frontotemporal and apparently large tumors, all six, or 100 per cent, had seizures. It is well known that convulsive attacks are more common with certain types of brain tumors than with others, more common with meningiomas and astrocytomas than with glioblastomas. Hence search was made for a reported series of meningiomas close to the motor pathways to ascertain whether the incidence of seizures was particularly high. Such proved to

TABLE III.

RELATION OF CEREBRAL LOCALIZATION TO CONVULSIONS.

Author.	Location.	No. of cases.	Per cent with convulsions.
Elsberg [15]	Temporal lobe (under surface)	15	80.0
Parker [10]	Frontal lobe	30	78.0
	Frontotemporal	6	100.0
	Parietal	21	47.6
	Frontoparietal	21	23.8
	Temporal	22	31.8
Kennedy [16]	Temporal	9	78.0
Cushing [17]	Temporal	59	34.0
Müller [18]	Frontal	164	37.2
Sargent [1]	Frontal	...	50.0
Gibbs [11]	Frontal	262	40.0
	Parietal	168	40.0
	Temporal	96	30.0
Groff [24]	Parietal (meningiomas)	34	76.4
	Frontal (meningiomas)	40	37.5
	Paracentral (meningiomas)	17	88.2
Voris [25]	Frontal	314	40.0 69 *

* Includes Jacksonian and petit mal attacks as well as grand mal.

be the case with Elsberg's 15 cases of meningiomas arising on the under surface of the temporal lobe, 12, or 80 per cent, having convulsions before operation. In 17 parasagittal meningiomas Groff [24] reports 15, or 88 per cent, with convulsions.

In connection with tumors, the high percentage of complicating seizures is not due to increased intracranial pressure. Series of cerebellar tumors with high intracranial pressure reveal very few cases of convulsions. (Stewart and Holmes,[20] 40 cases, no convulsions; Sachs and Furlow,[9] 247 cases, 4.8 per cent; Parker,[10]

71 posterior fossa cases, none; Groff,[24] 21 posterior fossa cases, none; Davis and Droegemueller,[22] 42 posterior fossa cases, 8 with convulsions.)

Reference must be made to the work of the Gibbs[19] on the convulsive threshold of various parts of the cat's brain. These investigators discovered that the electrical points of greatest excitability lay along a stretch suggesting a short fiber tract connecting the frontal cortex, thalamus and basal ganglia. These data conform with the foregoing clinical observations of the higher incidence of paroxysms in lesions of the frontal, parietal and temporal lobes than with lesions elsewhere in the brain. The location of brain lesions therefore has an important bearing on the occurrence of convulsions.

The frequent occurrence of seizures in certain brain neoplasms, at times as great as 80 per cent, suggests that genetic predisposition is an unlikely factor unless one assumes that this tendency to convulsions is linked to a hereditary disposition to tumors; a view for which no adequate substantiation is available. A similar assumption would be needed for the explanation of the traumatic epilepsies on a hereditary basis.

Since convulsive states occur in only 0.5 per cent or less of the general populace, the prevalence of epilepsy in those suffering from gross brain lesions is rather to be explained on the basis of cerebral insult than a genetic influence. Therefore the supposed infrequency of convulsions in brain lesions is not based on fact; to the contrary the incidence is rather high. Furthermore, the verification of a high incidence of epilepsy in brain disease may be advanced as an argument against an hereditary predisposition just as the supposed infrequent occurrence was said to be in favor of it.

From the data presented, although it cannot be stated that an anatomical localization for convulsive disorders has been demonstrated, enough has been revealed to infer that the location of brain lesions has much to do with the incidence of seizures. It is easy to see why lesions in the same location may or may not be accompanied by convulsions. In the first place, one cannot be sure of the localizations being exact, since the neural pathways involved are not known and brain lesions grossly, apparently alike and similarly located may still be quite dissimilar in terms of minute

cerebral anatomy. However, an even more important point arises from consideration of the nature of convulsions. Seizures are irritative symptoms as contrasted with "paralytic" symptoms based on loss of function. No lesion of the brain in and of itself is sufficient to produce convulsions. How else explain the intermittent character of the manifestations? A brain lesion is present continuously for some time, often days, months, years; still the attacks appear only occasionally with long free intervals. Hence another element, a precipitating factor, must be present. The latter may be absent in some cases even though the localization is exactly the same as another lesion productive of seizures. Some tumors close to the pyramidal tracts probably produce paralyses, thus inhibiting convulsions, destroying rather than stimulating the essential pathways. These and many other reasons might explain the failure of a specific anatomic brain lesion to induce these spells in 100 per cent of the cases. The incidence of seizures in anterior cerebral lesions is far too great to assume a hereditary predisposition unless we assume that the predisposition is a dominant Mendelian trait; a feature not borne out by the frequency of the syndrome.

More significant is the fact that certain methods for producing experimental convulsions are so universal in their capacity to cause seizures that a genetic influence in this connection need not be considered. An electric current applied to the motor cortex produces convulsions in all instances, and presumably the same can be said for convulsant drugs and cerebral anemia as convulsive measures.* When one adds the evidence from brain injuries and brain tumors, how much less necessary is it to assume a constitutional predisposition?

These facts do not *exclude* the possibility of an hereditary basis. They do serve to point out that one of the frequently preferred sets of data for a "predisposition" must be discarded and, further, that in the face of the frequency with which pathology of certain

*True, the threshold for these convulsant measures may vary from individual to individual, as they may presumably in the same individual from time to time. Still this is no proof of a genetic determinant for the differences in threshold. Many acquired factors may be responsible. No one thinks seriously of bringing in an hereditary predisposition to explain the fact that some persons respond to emetics by vomiting with a smaller dose of medicine than do other individuals.

restrictions (type and location) produces convulsions, the burden of proof must rest on those who contend that there is a genetic susceptibility to epilepsy.

From the foregoing, the following conclusions seem justified.

1. Old statistics indicating that convulsions are an infrequent occurrence in series of brain lesions, and therefore favoring an hereditary predisposition for epilepsy, must be revised in the light of more recent data.

2. Traumatic injuries to the brain (gunshot wounds) result in seizures in 20 per cent or more of the cases. In large series of brain tumors the incidence of convulsions is from 15 to 39 per cent.

3. When lesions of the anterior cerebral areas are considered, such as the frontal, parietal and temporal lobes, the incidence of convulsions is much higher. Brain tumors of this location are associated with seizures in from 30 to 80 per cent of the cases. Cerebral injuries manifest the same tendency when studied from the standpoint of localization.

4. The high incidence of the attacks with certain lesions of the brain invalidates the assumption of an hereditary predisposition to explain the occurrence of seizures in these cases. It places the burden of proof on those who contend that there is such a factor.

II. Familial Incidence of Seizures.—Studies on the family trees of epileptics purporting to show an increased incidence of convulsive disorders has been presented as evidence of an hereditary factor. The evidence may be evaluated from two standpoints. First, is there actually a familial preponderance, *i. e.*, are the statistical data adequate? Second, does an increased familial incidence necessarily indicate an hereditary influence? In the following discussion we expect to show that neither the first nor the second can be definitely answered in the affirmative.

The statistical reports on the frequency of epilepsy in families show no uniformity, hence the necessity for controls. The number of children similarly afflicted in families of epileptics is reported by different authors as high as 50 per cent and again by others as low as 0.29 per cent. Where the results vary so greatly, obviously one is at a loss to know how to evaluate the figures and profound skepticism is justifiable. In part differences are due to selection of material, definition of epilepsy, inclusion of infants dying in convulsions, and probably also to different statistical methods. In

view of the gross discrepancies from series to series and because of the lack of uniformity of the statistical approach, it is imperative that controlled series, and these alone, be considered. Although there are numerous studies on this subject we know of very few which have been performed on patients with "normal" controls. Those of Lennox and Cobb,[27] Stein [28] and Brain [29] are of this type, and they report an increased percentage of convulsive disorders in the families of epileptics, as contrasted to those of the controls. These studies call for detailed analysis.

Certain limitations in the controls raise doubt as to the validity of this material as a basis of comparison with the pathologic cases. In two similarly conducted investigations, those of Lennox and Cobb, and of Stein, the controls showed a difference between themselves of 500 per cent. Lennox and Cobb reported that 2.1 per cent of the immediate relatives of 1086 out-patient department epileptics were affected with a convulsive disorder, whereas this was true of only 0.26 per cent of the 250 normal controls. Stein's series of 1000 institutionalized cases at the Monson State Hospital and 1115 normal controls showed affliction in 3.7 per cent of the members of epileptic families and in 1.3 per cent of the members of control families. Although the incidence in the epileptic families in both groups was relatively not very different, 2.1 per cent and 3.7 per cent respectively, still there was a very marked difference in the control families, 0.26 per cent and 1.3 per cent; a difference of nearly 500 per cent where the figures should particularly coincide. Where such gross errors can creep in one must be wary of interpretations. It still remains true that in both studies the incidence of epilepsy was much greater in the patients' families as compared to the controls.

Discrepancies in the controls probably arose from the manner of selection and also from the means of obtaining information. The controls in the first series were nurses and medical students; in the second group there were included also teachers, doctors and social service workers. The validity of selecting controls from economic and social strata other than those of the patients is subject to question. Doctors, teachers, nurses and social service workers represent a select group. One might suspect that these individuals come from sheltered families where less of the environmental stresses contributory to epilepsy operate than among

the poorer hospital clientele. It is, for instance, much less likely that an epileptic father will be able to send his son through medical school than one not so handicapped. For these and probably many other reasons it is justifiable to insist on a series of controls from the same social stratum as the patients.

Information from the controls was obtained by *questionnaire,* but direct questioning was employed for the patients or accompanying guardians, frequently supplemented by subsequent reports from relatives secured by social workers. For statistical accuracy there should be the identical procedure for collecting data on the controls as on the patients. If direct questioning is the procedure with one, then the same should be utilized with the others; if accompanying guardians or absent relatives are consulted the same should be the approach for both patients and controls. Apparently such was not the case in these two studies. The contributions of Lennox and Cobb and of Stein cannot therefore be accepted as evidence of an increased familial frequency of epilepsy since the controls are selected and the method of collecting data varied in the epileptics and controls.

Brain conducted his study in a manner different from the foregoing investigators. He tabulated the number of epileptics and controls who had an epileptic relative. He personally interviewed 200 epileptics and 100 non-epileptic patients in the out-patient department of a nervous disease hospital. He recorded a "positive" family history in 28 per cent of the epileptic cases and in 10 per cent of the controls. Here we cannot make the criticism proffered against the previous studies. One may question the adequacy of using other nervous disease patients as controls. If there should be any hereditary illnesses in the control group the use of patients as controls would be invalid. However, a more cogent argument against accepting this report in favor of an hereditary factor in epilepsy follows.

Even were one to accept the foregoing figures as adequately controlled statistics, there would still be serious doubt as to the validity of the data. It is well to bear in mind that epileptics or their close relatives know the morbidity of their family trees better than do normal persons. Not uncommonly an epileptic patient tells us to what lengths he has gone to investigate the family history for this information. We know that many a distant relative would

find no occasion to mention to a healthy relative a convulsive disorder in himself, a child, or other member of the family, whereas he would readily divulge that information to a similarly afflicted one. Likewise a transient period of convulsions in a parent or sibling might not have come to the notice of a normal control, but would usually be known to an epileptic. Also the increased knowledge of the latter might lead to the inclusion of cases in the family tree with petit mal, syncopal attacks and other partial epileptic manifestations which might be lacking in the case of the control. Hence another factor detracting from this evidence favoring an hereditary predisposition in epilepsy may be the inability of controls to supply as accurate information as the patients themselves.

Before leaving the statistical evidence we should make reference to Paskind and Brown's [26] study of 342 children of 163 epileptics seen in private practice, only 0.29 per cent of whom had epilepsy. This incidence is little or no greater than the frequency of epilepsy in the general population and is therefore worthy of special comment. One would of course want to know until what age these children were followed, and even other details; yet this study, although not conclusive proof of the absence of an hereditary factor, serves to cast doubt on it.

Now we come to our second query, Does an increased familial incidence necessarily indicate an hereditary influence? *Familial predominance of an illness is by no means proof of its hereditary character.* Many acquired causative factors may be potent within the family group, for instance, an infective agent, a dietary deficiency, or injury due to birth through contracted pelvis or uniformly poor obstetrical care, all of which are accepted as having a causal relationship to epilepsy. In the pre-bacterial period the increased family incidence of tuberculosis might well have been accepted as evidence of the hereditary character of this disease. Because so many environmental factors enhance the familial occurrence of disease, such an increase cannot be accepted as proof of a genetic influence.

The foregoing discussion may be summarized as indicating that the studies on the familial occurrence of epilepsy cannot be accepted as unequivocal evidence of an hereditary predisposition.

1. An increased familial incidence has not been established as a fact, since (a) the controls in previously reported series were

not adequate from the standpoint of collection of data or selection of subjects, and (b) the specific family histories obtained from epileptic patients are more accurate and inclusive than those from normal persons.

2. Familial preponderance of a disease is not proof of its hereditary character.

Again, this exposition merely negates the existing evidence; it does not prove that heredity is not a factor.

III. Evidence from Study of Twins.—Twins present good material for the study of heredity in human diseases. Monozygotic twins, twins arising from one egg, have identical heredity, whereas dizygotic twins, twins arising from two eggs, have no greater hereditary similarity than other siblings—brothers or sisters. Therefore an inherited disease will appear in both members of afflicted identical twins and to a lesser degree in both members of dizygotic twins. If heredity is the sole etiologic factor in no instance of monozygotic twins would one twin be affected and the other not affected, whereas among the dizygotic twins the frequency of only one member being affected with the illness would be the same as that among siblings.

Because of these factors the study of epilepsy in twins has been undertaken by numerous investigators. Of recent times two studies have appeared which are outstanding because of the large number of cases analyzed. Rosanoff [30] reported both members affected with epilepsy in 52.2 per cent of the monozygotic twins and 10.7 per cent of the dizygotic twins. Conrad [31] collected statistics on 253 pairs of twins and reports concordant involvement in 66.8 per cent of the monozygotic twins as against only 3.1 per cent of the dizygotic twins. Conrad points out that if the idiopathic cases are separated from the symptomatic epilepsies the per cent of concordance rises from 66.6 per cent to 86.3 per cent. Both authors contend that the high concordance of illness in monozygotic twins and the lower concordance among dizygotic twins is evidence of an hereditary factor in epilepsy.

These data need to be analyzed to see whether any other explanation than an hereditary factor is possible for these highly suggestive figures. If it can be demonstrated that monozygotic twins are more nearly affected equally by some environmental or acquired factors than dizygotic twins, then inheritance cannot be

deduced solely from the foregoing statistics. That such is the case would appear to be indicated by the following considerations.

1. Monozygotic twins being more alike anatomically, physiologically and biologically than dizygotic twins would probably respond to generalized toxic or other noxious influences more uniformly with the same pathologic entity in both members than would dizygotic twins.*

2. Monozygotic twins have only one and the same placenta whereas dizygotic twins have two or separate placentæ. Hence placental abnormalities in one of dizygotic twins would be less

* This statement has caused some of our confreres to insist that since the similarity of monozygotic twins is due to like heredity therefore the similar response to environmental noxæ is evidence of the hereditary origin of the disorder. That such is not the case we believe can be shown by the following hypothetical case. Let us take two sets of twins, one monozygotic and the other dizygotic. Let us assume that both were subjected to the same type of environmental handicap, a contracted bony pelvis. Let us assume further that the monozygotic twins had egg-shaped heads and that in the dizygotic twins, one had an egg-shaped head and the other a spherical head. Neither one of the heads is pathologic and both can pass through a normal birth canal. If the contracted canal can be readily passed by the spherical skull and not by the egg-shaped ones, then we may have cerebral birth trauma in both monozygotic twins and in only one of the dizygotic twins as a background for a convulsive disorder. Now one might insist that this identity of pathologic response in both monozygotic twins proves the hereditary nature of the disorder, since both heads were egg-shaped in the monozygotic twins because of certain genes transmitting this character. The hereditary basis for the egg-shaped head cannot be denied. However, the egg-shaped head cannot be considered the cause of the epilepsy, but the emphasis must be placed on the contracted pelvis as the responsible agent. The egg-shaped head is not pathologic. In a normal mother or at an earlier period in the pregnancy no abnormality in the birth process would have resulted. Further in another woman with a contracted pelvis, the spherical head might be the one which could not pass through readily, whereas the egg-shapped skull would. We have here an example of a defective environmental factor which in the face of normal heredity causes a greater concordance of pathology in monozygotic than in dizygotic twins. The only point we wish to make, therefore, is that features in twins ordinarily not pathological may be responsible for differences in response to external stimuli; that monozygotic twins, because they are more nearly alike, will respond similarly and dizygotic twins, not being alike, will more frequently respond differently. The extent to which this may be responsible for the greater concordance of pathologic disturbances in monozygotic as compared to dizygotic twins cannot be stated, but it must be definitely considered.

likely to affect both members than in the case of the monozygotic twins.

3. Finally, it seems reasonable that there may be many other intrauterine acquired factors with which we are not acquainted which may possibly affect the concordance of twin involvement in the same direction as above.

4. Postnatal factors likewise would be expected in some instances to affect monozygotic twins more uniformly than dizygotic twins. Again the sameness of monozygotic twins so frequently as to biological characteristics in comparison with the lesser similarity in dizygotic twins would call for a more uniform response in the former to infections, toxins, and dietary deficiencies.

5. If it could be shown that identical twins have more nearly the same likes and dislikes, the same behavior, than dizygotic twins, then it is altogether possible that the greater selection of the same environmental exposures of monozygotic as compared to dizygotic twins would in itself be responsible in part for the difference in the incidence of concordance in both types of twins.

Because of these considerations we think it safe to conclude that environmental or acquired factors may play a rôle as to the greater concordance of epilepsy in monozygotic twins than in dizygotic twins. Until it can be shown that these factors are inadequate to explain the difference in the two groups it must be admitted that the evidence presented is not proof of an hereditary factor.

Besides recognizing that the results from twins studies may be explainable by environmental or acquired factors and not necessarily genetic influences, it may be of value to investigate the validity of the method for this type of study. Twin studies for the detection or verification of hereditary origin are of greatest value where the entity is (1) *readily recognized and* (2) *solely and exclusively a unitary genetic character.* The epileptic predisposition, however, is neither a sharply limited, readily identifiable unit nor solely of hereditary origin, since some acquired etiologic factors are universally admitted. If epilepsy as such was being transmitted, clinical recognition would be simple. Since it is predisposition to epilepsy which is suspected of being transmitted by the genes, the identification is by no means easy or always possible. Difficulty arises from the fact that there is no uniformity of definition, no uniformity of opinion as to the number of indi-

viduals with the predisposition who subsequently develop seizures or under what circumstances, and no uniformity of opinion as to whether there must be a predisposition in *every* epileptic and, if not (as seems altogether probable), then in what cases it is present. Despite the intangible character of the concept of epileptic predisposition, this is the entity which must be measured by statistical studies.

Then too, the epileptic predisposition is not an hereditary unit character. Many acquired factors are known to be predisposing agents, capable for instance of lowering the convulsive threshold. In the first section of this paper, the discussion referred to the causes of epilepsy as being predisposing or precipitating. This would place the organic lesions of the brain in the category of predisposing factors, some of which may be definitely hereditary *e. g.*, aplasias. Besides these we know of certain physiologic states which may serve in the same capacity, such as hydration, anoxemia, and alkalosis. Therefore epileptic predisposition is obviously not a sole genetic unit, but if there is a universal hereditary component it is only one of many contributory causes. This must of necessity complicate the statistical procedure. Where the entity to be studied is so poorly identifiable and not exclusively a *unit* of hereditary or environmental origin, then the validity of the twin method may be seriously questioned as an adequate technic for determining genetic origins. At the present time the method is based on two assumptions, (1) that the number of cases with the predisposition which becomes manifest as seizures will be the same in identical and dizygotic twins and (2) that the hereditary factors alone will be able to explain the greater concordance in single-egg twins. The former assumption is not necessarily so and the latter we have indicated may not be so since environmental factors may well account for greater concordance of pathology in monozygotic than in dyzygotic twins.

So far we have concerned ourselves with theoretical formulations. We shall now call attention to two factual items which we believe show that the foregoing objections voiced have not been without warrant. In Rosanoff's series in 10.7 per cent of the dizygotic twins both members had epilepsy, whereas in Conrad's cases only 3 per cent of the dizygotic twins were so affected. Again it must be stated that in both series the concordance in the

identical twins was much higher, in Rosanoff's series approximately 4.9 and in Conrad's series 21 times as great as in the dizygotic twins if all cases are included and still greater if only the idiopathic cases are considered. The fact still remains that if the method gave an accurate evaluation of the data, such discrepancies in the two studies should not exist. Such gross differences for a method based on the same principles calls for caution as to its positive application.

Of more significance for our thesis is the fact that a study of twins affected with tuberculosis gave the same results as obtained in epilepsy. Diehl and v. Verschuer [32] found concordant involvement with tuberculosis in 70 per cent of monozygotic twins and in 25 per cent of dizygotic twins. This does not differ greatly from Rosanoff's series of epileptics with figures of 52.2 per cent and 10.7 per cent respectively in the monozygotic and dizygotic twins. If the evidence from twin studies is accepted as proof of an hereditary factor in epilepsy, then the same obtains for such a factor in tuberculosis. It seems more probable to us that the environmental influences bearing upon monozygotic and dizygotic twins before, during and shortly after birth may well be responsible rather than differences in the germplasm. At least it reduces the problem to the point where the twin evidence for an hereditary factor in epilepsy is no greater than the same evidence in tuberculosis. It will probably be stated that there is an hereditary predisposition in tuberculosis and hence the data are still valid for epilepsy. Our answer is that we are interested in heredity from a practical etiologic point of view; namely only as a palpable, relatively significant factor. Tuberculosis cannot be caused without the tubercle bacillus. True we recognize such predisposing conditions as malnutrition, lack of sunlight and fresh air, and overexposure to cold and dampness. And one cannot say that there is no hereditary element involved. Nevertheless, the tubercle bacillus remains the major etiologic agent. From a practical standpoint we are not interested in heredity if it operates as a relatively insignificant contributory factor. Certainly the significance of genetic origins in tuberculosis has been relegated to quite a different place in the etiology since the discovery of the tubercle bacillus as compared to prior times. The same would be true if tomorrow some etiologic agent as definite as the tubercle bacillus

was discovered for the so-called cases of idiopathic epilepsy. The twin data apparently do not exclude such a possibility. The differences in the studies of Rosanoff and Conrad and the similarity of the twin statistics for tuberculosis and epilepsy indicate that doubt as to the adequacy of the twin method for our problem is not without justification.

In conclusion, we would like to point out that

1. Studies of twins affected with epilepsy point to a greater concordance (involvement of both members of the twin pair) in monozygotic than dizygotic twins. This has usually been accepted as evidence of a hereditary predisposition to epilepsy.

2. Environmental factors have been suggested which may be responsible for the differences noted in the two twin groups.

3. The validity of the twin approach is questioned since epilepsy is recognized as a syndrome due in known cases to many different causes and not to a single unitary factor. The application of this method is further limited by the absence of a procedure for detecting the "predisposition" in so-called latent cases.

4. The same differences between monozygotic and dizygotic twins with epilepsy can be demonstrated for tuberculosis.

Summary and Conclusions.

The major evidence favoring an hereditary predisposition in epilepsy is reviewed.

1. The old dictum that epilepsy complicates brain lesions infrequently has been shown to be inaccurate in the face of modern statistics. To the contrary, convulsions are a frequent manifestation in cases of brain injury and tumors, and particularly so if the lesions are in the anterior portions of the cerebral hemispheres. These facts do away with the necessity of assuming an hereditary predisposition to explain why certain lesions of the brain are associated with convulsions and others not.

2. Evidence arising from the familial incidence of epilepsy is called in question on the grounds of statistical accuracy. Controls are shown to be selected from different social strata and information secured from them in a manner different from that in the patients. Attention is called to the fact that epileptics know their

family trees much better than do normal controls. Finally it is indicated that many environmental factors may produce an increased familial incidence of a disease, *e. g.*, tuberculosis, and hence, even if definitely established as a fact, an increased familial incidence of epilepsy would not in itself be conclusive proof of its hereditary origin.

3. Twin studies in epilepsy show a greater degree of involvement of both members of monozygotic twins than dizygotic twins. This concordant preponderance in the identical twins may be due to acquired factors as well as genetic influences and hence is not proof for an hereditary etiologic factor. The validity of the method of twin study is questioned in the face of so difficultly identifiable an entity as epileptic predisposition and because heredity is only one of a number of other factors instead of the sole etiologic agent. Here too in an acquired illness such as tuberculosis the statistics from twin studies are the same as those in epilepsy.

4. It is definitely stipulated that the entire discussion herein of the evidence for an hereditary predisposition is not proof of the absence of such a factor. The contention is fostered that the present day evidence is inadequate to establish heredity as a significant contributing etiologic element. The burden of proof still must rest on those who contend that heredity is of importance in this pathologic condition.

5. Finally, although no definite or direct evidence has been advanced to show that there is no hereditary predisposition, attention might be called to the field of experimental convulsions. In the laboratory the universal efficacy of various experimental procedures such as electric current, convulsant drugs, and methods inducing cerebral anemia, tends to indicate that hereditary predisposition is not a necessary element in the production of seizures and that if it exists at all it must be present as a minor factor.

BIBLIOGRAPHY.

1. Sargent, P.: Some observations on epilepsy. Brain, 44: 312, 1921
2. Wagstaffe, W. W.: The incidence of traumatic epilepsy after gunshot wounds of the head. Lancet, 2: 861, Oct. 27, 1928.
3. Frazier, C. H., and Ingham, S. D..: A review of the effects of gunshot wounds of the head. Arch. Neurol. and Psychiat., 3: 17, Jan., 1920.

4. Steinthal, K., and Nagel, H.: Die Leistungsfähigkeit im bürgerlicker Beruf nach Hirnschüssen mit besonderer Berucksichtigung der traumatischen Epilepsie. Beitr. z. klin. Chir., 137: 361, 1926.
5. Quoted in Schou, H.: Trauma capitis and epilepsy. Acta psychiat. et neurol., 8: 75, 1933.
6. Rawling, L. B.: Gunshot wounds of head. Brit. J. Surg., 10: 93, July, 1922.
7. Schürer-Waldheim: Die chirurgisch Behandlung der Epilepsie. Wien. med. Wchnschr., 84: 1180 and 1239, 1934.
8. Dowman, C. E., and Smith, W. A.: Brain tumors: Analysis of one hundred cases. J. A. M. A., 90: 321, Jan. 28, 1928.
9. Sachs, E., and Furlow, L. T.: The significance of convulsions in the diagnosis of brain tumor. J. Missouri State M. A., 33: 121, April, 1936.
10. Parker, H. L.: Epileptiform convulsions: The incidence of attacks in cases of intracranial tumor. Arch. Neurol. and Psychiat., 23: 1032, May, 1930.
11. Gibbs, F. A.: Frequency with which tumors in various parts of the brain produce certain symptoms. Arch. Neurol. and Psychiat., 28: 969, Nov. 1932.
12. Spratling, W. P.: Epilepsy and its treatment. Phila. and N. Y., W. B. Saunders Co., 1904.
13. Holt, L. E., and Howland, J.: Diseases of infancy and childhood. New York, D. Appleton and Co., 1922.
14. Ford, F. R.: Cerebral birth injuries and their results. Medicine, 5: 121-194, May 1926.
15. Elsberg, C. A.: The meningeal fibroblastomas on the under surface of the temporal lobe, and their surgical treatment. Bull. Neurol. Inst. N. Y., 2: 95, March, 1932.
16. Kennedy, F.: The Symptomatology of temporosphenoidal tumors. Arch. Int. Med., 8: 317, Sept. 1911.
17. Cushing, H.: Distortions of the visual fields in cases of brain tumor: The field defects produced by temporal lobe lesions. Brain, 44: 341, 1922.
18. Müller, E.: Zur symptomatologie und diagnostik der geschwülste des stirnhirns. Deutsche Ztschr. f. Nervenh., 22: 375, 1902.
19. Gibbs, F. A., and Gibbs, E. L.: The convulsive threshold of various parts of the cat's brain. Arch. Neurol. and Psychiat., 35: 109, Jan. 1936.
20. Stewart, T. G., and Holmes, G.: Symptomatology of cerebellar tumors: a study of forty cases. Brain, 27: 522, 1904.
21. Baumeyer, S.: Traumatic epilepsy in ex-service men. J. A. M. A., 106: 934, 1936.
22. Davis, L., and Droegemueller, W.: The surgical treatment of epileptiform seizures. Ann. Surg., 103: 669, 1936.
23. List, C. F.: Epileptiform attacks in cases of glioma of the cerebral hemispheres. Arch. Neurol., and Psychiat., 35: 323, 1936.

24. Groff, R. A.: The meningioma as a cause of epilepsy. Ann. Surg., 101: 167, 1935.
25. Voris, H. C.: Tumors of the Frontal Lobe. J. A. M. A., 104: 93, 1935.
26. Paskind, H. A., and Brown, M.: Frequency of epilepsy in offspring of persons with epilepsy. With special reference to differences between institutional and extramural patients. Arch. Neurol. and Psychiat., 36: 1044, November, 1936.
27. Lennox, W., and Cobb, S.: "Epilepsy," Williams and Wilkins Press, Baltimore, p. 143, 1928.
28. Stein, C.: Hereditary factors in epilepsy. Am. J. Psychiat., 89: 989, 1932.
29. Brain, W. R.: The inheritance of epilepsy. Quarterly Journal of Medicine., 19: 299, 1926.
30. Rosanoff, A. J., Handy, L. M., and Rosanoff, I. A.: Etiology of epilepsy with special reference to its occurrence in twins. Arch. Neurol. and Psychiat., 31: 1165, June 1934.
31. Conrad, K.: Erbanlage und epilepsie. Untersuchungen an einer serie von 253 zwillingspaaren. Ztschr. f. d. ges. Neurol. u. Psychiat., 153: 271, 1935.
32. Diehl K. u. v. Verschuer, O.: Zwillingstuberkulose. Zwillingsforschung und erbliche Tuberkulosedisposition. Gustav Fischer, June, 1933.

DISCUSSION.

DR. AARON J. ROSANOFF (Los Angeles, Calif.).—Dr. Ziskind endeavors to disprove the existence of a hereditary factor in the etiology of epilepsy by attacking, separately, in the manner of Napoleonic military tactics, the evidence based on clinical statistics, familial studies, and observations in monozygotic and dizygotic twins. The evidence yielded by each of these groups of data is, in itself, of course inconclusive. The evidence as a whole, however, points so consistently to the existence of a hereditary factor, that it would seem to amount to something more than a mere summation of data; it would seem rather to present, by the harmony of the data obtained by various modes of investigation and from various sources, a definite preponderance for, rather than against, heredity in the etiology.

Dr. Ziskind does not deny this; he merely states that the proof thus far submitted is still inconclusive; and with that we may well agree.

In the course of his discussion he presents no new data that would help us toward a more precise evaluation of the possible hereditary factors in etiology. The paper is no contribution to our knowledge of the subject. It would seem to me that one who has such good suggestions for improved ways of gathering, analyzing, and presenting evidence pertaining to hereditary factors in etiology, would do better if, instead of pointing out the weaknesses of older studies, he would but roll up his sleeves, go to work, and contribute something that might really help to settle the controversy.

CHAIRMAN LENNOX.—There are one or two points in which my name was brought in and on which I ought to perhaps give some defense. First,

this matter of familial incidence of seizures among the relatives of the patients with seizures. In the control data which we have secured now after a number of years of questioning, we have now a record of nearly 1000 normal individuals with 5000 near relatives, and the percentage there is 0.5 per cent, which is just about the figure gained in the draft; that is 0.5 per cent of the population with seizures. This is not just data of students and nurses that we questioned, but we have gone into the medical wards and questioned the patients there, which makes a comparable group to the epileptic patients who are questioned, using the same blanks.

This figure, 0.5 per cent, is about one-sixth of the figure for the relatives of the epileptics, and that sixfold difference is a bit hard to lop off. It is especially hard to lop off when we consider the group of the epileptic patients who are the symptomatic patients, that is, the traumatic epileptics; and we find that the family history of seizures in this group runs about four times the incidence of seizures in the general population as against about seven times for the so-called idiopathic group.

Then the matter of twins. It seems to me that the data on tuberculosis are very apt. They show there is high concordance in monozygotic twins with tuberculosis. There is a hereditary factor as well as a germ factor in the etiology of tuberculosis. As a matter of fact, that same thing can be said for diabetes. If Dr. Ziskind means to say that the hereditary factor is not as great as it has been commonly preached from the house tops, then I quite agree with him. If, on the other hand, he says, as I think he does, that it is of no account, then I thoroughly disagree with him.

DR. ZISKIND.—I agree with Dr. Rosanoff that a review of this type is not as definite a contribution as a positive investigation to prove an issue. We thought seriously about this. In fact our study of the literature was for the purpose of orientation in such a project. We have investigated about 600 epileptics in the last 6 years at the Los Angeles County General Hospital but hesitated to repeat studies already done, probably yielding the same type of results with the same deficiencies. However, we must insist that it is of value to point out the invalidity of certain concepts generally accepted, since this clears the way for further work.

The crux of the argument rests in whether environmental or acquired etiologic factors can produce an increased familial incidence of a disease process and also a preponderant concordant involvement in monozygotic over dizygotic twins. We have contended that such is the case; that acquired as well as hereditary factors may produce this result. Since the discussants have not denied this, may we assume that they agree? If such is the case, what right have we to decide that the familial studies and observations in the twins are greater proof of the hereditary factor than of the environmental or acquired one? Would it not be better to take an agnostic attitude, to admit that the question is still unsettled?

We did not say that Dr. Lennox's statistics do not show an increased familial incidence, but merely wish to call attention to the difference of 500

per cent obtained in the controls by two different investigators using his procedure. Dr. Lennox's larger series diminishes this difference, but there is still sufficient variance to cast doubt on the validity of the method. That the traumatic cases show a lesser familial incidence than the idiopathic cases is what one might expect and is by no means proof of a hereditary factor. It may indicate merely that among the unknown etiologic factors causing the "idiopathic" epilepsies there are some which have a greater tendency to familial incidence than do head injuries. Infectious processes and dietary deficiencies (if etiologic factors) occurring in the idiopathic group could give rise to such a difference as cited between the traumatic and idiopathic cases.

Our review does not disprove the presence of a hereditary factor, but the evidence favoring such is inadequate. That leaves the question open. And since all of us demand some proof for the presence of any etiologic agent, it places the matter of heredity in relation to epilepsy in a far different light than that usually held. When the acquired causative factors in epilepsy are as well known and evaluated as those in tuberculosis, it may well be that heredity in regard to the former will be relegated to as unimportant a position as it holds today in the minds of most clinicians for tuberculosis.

AN HISTORICAL VIEW OF SOMATIC TREATMENT IN PSYCHIATRY.*

By OSKAR DIETHELM, M. D., New York City.

During the whole development of scientific psychiatry, which began during the latter part of the eighteenth century, two exclusive conceptions have dominated medical thinking and treatment. On the one hand mental illnesses were conceived as having a physical basis, either localized in the brain or other diseased organs affecting the brain. On the other hand a purely psychic basis was maintained. A relatively small group tried to do justice to both concepts. The hope that these diverse theories might become united constructively with the development of a psychobiological psychiatry has not fully materialized as yet. The concept of psycho-physical parallelism has become replaced by psychobiological integration, but this fundamental change has not become as widespread or thorough as might seem from listening to psychiatric discussions. Too much lip service is given to the concepts of integration, disintegration, and reintegration. They are used with what resembles metaphysical rather than scientific elaboration, and behind these terms is recognizable the old separation of mental and physical.

It is deplorable that therapeutic activities are guided to a large extent by empiricism, *i. e.*, dependence on observation alone, without due regard to science and theory. Scientific medical thinking does not disapprove of making use of isolated clinical observations for therapeutic deductions but demands that such observations be critically investigated and generalizations avoided. Experimental controls are made an essential safeguard.

In 1820 Georget,[1] one of the most scientific psychiatrists of modern times, discussed the prevalent empirical treatment with criticism which is still valid today. He stressed the fact that the

* Read at the ninety-fourth annual meeting of The American Psychiatric Association, San Francisco, Calif., June 6-10, 1938.

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physician cannot ascertain underlying somatic changes in his patient, and postmortem control is rarely available, as most types of mental disorders are not fatal. He stated further that it is often difficult or impossible to establish an exact connection between the mental symptoms and the nature of the organic changes which produce them. The absence of specific symptoms forces the physician to do nothing or to act empirically. Georget claimed that nothing was known at this time of the action of drugs which affect primarily the brain. It was only possible to deduce or guess the effect on the brain and on mental disorders of remedies which were directed to the alimentary or cardiovascular systems. This scientific warning was heeded little by psychiatrists of his time when plunging into cold water was still widely advocated. This universally accepted treatment was based on Van Helmont's story in 1707 that a Dutch carpenter was cured accidentally in such a way from insanity. Georget's criticism is still appropriate today.

Other ill considered therapeutic activities link up with general biological and medical developments. The physiological theories and conceptions of each period are made the basis for therapeutic procedures, frequently with utter disregard for the statements of more careful observers. Too inclusive and often phantastic theories of mental disorders are apparently the inevitable results of uncritical elaborations. It is unfair to speak, as is customary, of the barbaric treatment of mental patients at the beginning of the nineteenth century. The physicians who proposed and used the swivel chair of Cox or the tranquillizer of Rush to produce nausea and resulting fatigue and sleep were probably as humanitarian as the psychiatrists of today. A statement of Burrows' [2] in 1828 in defense of gyration was that "any remedy that invites sleep acts more beneficially on the disturbed functions of the brain than narcotics of any kind, though producing that effect." The same reasoning is now the basis of recommending some hydrotherapeutic procedures, with none too clear physiological explanation of cerebral circulation. French psychiatrists of today claim that the swivel chair represents an attempt to cure through provocation of a "vagotonic crisis." [3] The empiricism of the eighteenth century becomes, according to them, supported by the neurophysiological research of the twentieth century. With the increased knowledge of sensory reactions, the hasty notion that psychotic

patients are insensible to heat and cold, and the resulting extraordinary therapeutic exposure to excessive temperature changes, was abandoned. Bloodletting, another widespread treatment of the eighteenth century, was considered indicated by the condition of the pulse. Pargeter [4] (1791) objected to this indication because the pulse was too much influenced by emotions of hope and fear and various physiological factors. He advocated bleeding to mitigate morbid heat and "evident congestion about the membranes of the cranium and brain." Copious bleeding was supposed to relieve the increased blood supply in the brain, which was thought to cause excitements. The observation that hemoptysis and hemorrhoidal bleeding [5] have been observed to precede convalescence, were other facts mentioned in support of this therapeutic procedure. Pinel [6] connected causally occurrences of bloodletting and later deterioration in schizophrenics and condemned, therefore, this therapeutic procedure. With Broussais' [7] theory of cerebral irritation (1828) a physiological basis was claimed for mental disorder which supported somatic procedures and offered justification for time-honored bloodletting, various types of hydrotherapy, skin secretion, and purgation. Striking individual observations had previously often been the empirical basis for these treatments. Pinel's and Esquirol's none too critical objections to bleeding were overruled by Ellis [8] in 1838 when the somaticists again became leading in English psychiatry. From his postmortem observations, Ellis felt justified in stating emphatically that increased sanguineous action takes place on the commencement of insanity. In a modified form, according to the medical thought of the time, venesection and especially local bleeding by use of leeches and cupping were recommended in the textbooks of Dagonet [9] (1862) and Griesinger [10] (1871).

It is of interest to note the avidity with which psychiatrists have reached for therapeutically stirring discoveries in the general field of medicine. It is an expression of therapeutic hopelessness in a naively hopeful physician. A telling example is offered by the use of etherization, prevalent in this country, where the anesthetic possibilities of ether had been discovered in 1846. Although advocated highly from 1849 to about 1860, etherization was of only transient significance in psychiatry. In America it was found valuable in acute excitements and depressive agitations, especially

with marked fear and outbursts of panic; while the French psychiatrists preferred it in demented schizophrenics.

Many pharmacodynamic treatments have been proposed on an empirical basis or defended by current physiological theories, usually applied with considerable enthusiasm but only occasionally over a period of many years. Sedatives have been found useful to alleviate irritation of the brain or to offer rest from worries by inducing sleep. At other times the change in cerebral circulation was considered the essential factor, and camphor and digitalis therefore highly praised as specific sedatives in mental disorders. The nauseating effect of mechanical gyration, considered as a wholesome sedative, was the contribution of the psychiatrists at the end of the eighteenth century; and apomorphine hypodermic administration was a like contribution at the end of the nineteenth century. Rush [11] stated that gyration "opposes the impetus of the blood towards the brain, it lessens muscular action everywhere, and reduces the force and frequency of the pulse," evacuates bowels and stomach, and produces sleep. The accompanying vomiting and nausea he believed to exert a strong and beneficial influence on the circulation of the brain. All these means were supposed to tend to restore the balance between the nervous and vascular systems which had become deranged in a psychosis. Whether lasting improvement in an excitement under such therapeutic administrations was due to the physiological mechanism or to the deterring effect of fear of repetition of these dreadful experiences was discussed with unbending tenacity by those who believed in somatic etiology on the one side, and those who defended the importance of psychic causes on the other. Treatment by the tranquillizer was recommended not only to make violent patients tractable and kind but also to interrupt "morbid association of ideas." The dread of being exposed to this machine again made patients alive to everything around them, and thus justified the use of the swivel chair in autistic and negativistic conditions.

An interesting example of the therapeutic use of insufficiently understood drugs and the accompanying danger is found in 1872 when Crichton Brown [12] strongly recommended the use of conium (hemlock) in excitements. He stated that the rapid movements in a motor excitement tend, by their nature and physiological concomitants, to excite the cerebral hemispheres and involve the

mental functions, leading to emotional stimulation which again stimulates muscular exercise. This seemed supported by the histological changes in the striate body which the author found in such patients. Believing that conium affects primarily the motor centers, which to him was the striate body, he urged its use in acute manic and schizophrenic excitements and also in marked agitated depressions. The true pharmacological action was found a few years later when it was demonstrated that conium causes partial paralysis of the skeletal muscle nerve endings, without any action of importance on the central nervous system. The desired effect of languor and drowsiness was of a serious toxic nature. Another example of uncritical thinking is the recommendation of the use of quinine in cases "of the recurrent form of insanity" in the middle of the nineteenth century. The proponents were influenced by the old theory that the excited patient has increased heat production and by the observation that quinine proved to be efficient in controlling recurrent fever. Many sedatives and narcotics have been recommended on similar mistaken grounds. It also might be wise to keep in mind that we do not only need much information on the pharmacodynamic and toxic nature of drugs we use but also on psychobiological reactions of individual patients who suffer from various personality disorders.

The desire to use the toxic effects of drugs to force a psychological reversal has been alluring to physicians since medieval times. In the last 50 years such attempts have been made with opium. Ranney [13] writes: "The first effect of opium is to allay the passions, not only by lessening directly the most violent anger and poignant grief, but also by occupying the attention with fanciful and pleasant imagery, tending to induce cheerfulness and contentment. Hyoscyamus, on the contrary, is supposed to arouse anger and jealousy, while belladonna, in large doses, occasions gloomy thoughts and dejection of mind. Stramonium affects the senses primarily, and, in moderate quantities, disposes to convulsive merriment. . . . . If this be granted, it necessarily follows that with a knowledge of the change produced by this class of remedies on the different faculties of the mind, a proper selection for the individual case must be attended with good results." It is hardly necessary to mention the therapeutic uselessness and danger of such medication. In more recent years the euphoric effect of acute

bromide intoxication has been advocated to change deep depressions into mild elations. Cannabis indica was claimed to increase the activity of the senses and its use is said to have been followed by rapid improvement in demented patients. In recent years mescal has been tried on quite an analogous basis.

While at the end of the eighteenth century, under the influence of Stahl's doctrines, the excited patient was starved "to relax the system and derive blood from the brain," a full diet with a supply of meat rich in fat was urged nearly a hundred years later to counteract the anæmic state of the brain, due to high excitement (Ranney). It was thought that a similar diet in demented patients would enrich the blood, stimulate the brain and arouse its dormant excitability, and that phosphates of iron would furnish the necessary amount of phosphorus for generating the nervous force in the brain. With physiological and biochemical advances of recent years our understanding of diet now seems to develop along sound lines.

In 1880 Beard [14] described neurasthenia, "a chronic, functional disease of the nervous system, the basis of which is impoverishment of nervous force, waste of nerve-tissue in excess of repair," the symptoms "the result of reflex irritations." Milk had just been found valuable as an exclusive diet in dyspeptic states, anemia, and diabetes; and it was prescribed by Beard and Mitchell [15] in the treatment of neurasthenia. It was not surprising that massage and electricity would again come into use at this time when electrophysiology was at its height, just as electrotherapy in psychoses had been widely adovcated at the end of the eighteenth century when electricity was first applied to medical treatment. Seclusion and rest, which had been advocated to diminish the sensory excitability in excitement a hundred years previously, were then found justified from the point of view of neurophysiological advances. Rest versus activity, separation versus group life, have remained controversial topics.

The functioning and malfunctioning of the gastrointestinal tract has for centuries been of great interest in the treatment of mental disorders. According to general medical knowledge and prejudices, hellebore, various cathartics, emetics, bowel resections and colonic irrigations have been and are still used, but rarely with an attempt even to determine what is actually happening in the body. The

physicians who advocate these remedies offer an explanation in the somewhat mythical terms of toxic factors rather than factual formulations. In this field the step from Pinel to twentieth century psychiatry is not far.

Focal infections have been considered for a long time. In the middle of the nineteenth century extractions of carious teeth were occasionally found to terminate excitements. It remained for the twentieth century, however, to let surgical overactivity take a lead in psychiatric treatment. A healthy sobering has now taken place, and the psychiatrist is guided by improved diagnostic means.

Endocrine treatment was successful in cretinism and myxedema, but there were no sufficient indications to justify its application to the treatment of schizophrenia. The reasons suggested were that deteriorated schizophrenic patients frequently looked like myxedematous cases; or that definite histopathological findings occurred in the sexual glands of these patients; or that the promises of Abderhalden's physiological method pointed to an endocrine disorder. The old observation that febrile diseases led to improvement in psychoses induced the psychiatrist of a hundred years ago to depend on setons and similar means to produce abscesses. This led Wagner-Jauregg to undertake a careful and systematic study. For over a period of 20 years he used various means which culminated in the successful malarial treatment of general paresis. In other psychoses this approach has not led to success.

The frequently narrow and unbending attitude of those psychiatrists who believed in the importance of psychic factors did not help to put somatic treatment in its correct place. Pinel's intolerant and shortsighted point of view destroyed, partially and temporarily, the influence of the somaticists at the beginning of the nineteenth century. At the same time Samuel Tuke [16] was more reserved in his criticism and was willing to use somatic treatment whenever indications seemed to exist. In German psychiatry a bitter fight between the two groups persisted for years. In this country the somaticists kept a stronghold on psychiatry throughout the last century. Essential psychotherapy was not able to develop, although Mesmer had laid the foundation for it in 1779.[17] Heinroth's books,[18] which have often been ridiculed and condemned, emphasized theoretical and practical points which at the end of the century began to be restated. His theory of the importance of guilt and

of the patient's responsibility with regard to his actions and resulting personality disorders are easily recognizable in some of the modern psychodynamic thinking. With the development of genuinely psychobiological psychiatry in the twentieth century the schism between psychic and somatic treatment has been overcome. The psychiatrist began to realize that in any somatic treatment the psychological factors must be investigated and evaluated. In psychotherapy the resulting somatic reactions are important. It is now accepted that "in a disturbance in whatever level of integration, one must examine carefully the other levels of integration and consider possible interrelations and influences. In other words, one always has to consider the total organism" (Diethelm).[19] This implies that whatever is accessible to such procedures should be treated chemically and physiologically, but always with frank recognition that the basis for treatment is the understanding of the patient's personality. This modern psychobiological concept was already present in some of the writings of German authors at the beginning of the nineteenth century, and it became an important consideration through the influence of Carus [20] in the middle of the century. This valuable conception did not obtain a leading influence, however, until it was presented in its modern form by Adolf Meyer. Any present day somatic treatment needs to be evaluated from a psychobiological point of view. It has become essential to determine what psychological factors might be involved, and it seems no longer justifiable to infer from the success of a somatic interference that a structural basis for an illness is demonstrated.

Among present day somatic treatments some have proven to be of considerable value, or at least interest, and deserve special discussion. Some were arrived at primarily on a theoretical basis, while others resulted from clinical observations. The prolonged sleep treatment with trional was first suggested for the management of confusional excitements in 1901 (Wolff). Epifanio took it up again in 1916, using phenobarbital, because he felt that sleep was a function of rest which would bring pathological thinking to a standstill and interrupt an involved and tiresome thought process. In 1922 Kläsi [21] introduced prolonged sleep treatment by the means of somnifaine, and later dial (diallyl-barbituric acid), based on the hypothesis that inflammatory processes healed through anesthesia

by a discontinuance of the vicious circle between stimulus, insufficient blood supply, increased transudation and increase of the stimuli. He claimed that such anesthesia in a schizophrenic excitement would be quieting and heal the symptoms which are caused by retroceptive and central irritation. His second point was based on the observation that schizophrenic illnesses improve after febrile diseases or artificial fever. He explained this observation by the influence of the improved rapport between patient and physician due to the physical illness and the need for help. This latter aspect has been increasingly emphasized by Kläsi, who abandoned the physiological explanation a few years later and paid special attention to carefully planned psychotherapy, especially in connection with the awakening from the prolonged sleep and the reorientation to the hospital and outside activities. The biochemists [22] suggest that oxidation of the brain is considerably disturbed in various psychoses. A state of anoxemia is produced through the sleep treatment and is considered a significant therapeutic factor.

A similar explanation seems to play a rôle in various other therapeutic attempts, the best known of which is the so-called insulin treatment of schizophrenic disorders. In psychiatric treatment, insulin was used first to improve depressions by a general metabolic change and also tried for stimulation of the appetite in depressive and negativistic conditions, then for the treatment of delirium tremens, later to alleviate the disturbing symptoms during withdrawal of morphine, and finally to cure schizophrenic disorders. The theoretical basis for these various therapeutic applications varied considerably. The change in sugar tolerance curves in depressions was not accompanied by clinical improvement, just as a hundred years ago the decrease of pulse rate in agitated depressions by the administration of digitalis did not change the psychosis. The hunger due to hypoglycemia proved to be inadequate to take care of the appetite problems of the depressed patient or to overcome the negativistic refusal of food. In delirium tremens the expected beneficial influence on disordered functions of the liver did not materialize. The symptoms of withdrawal of morphine are alleviated greatly by insulin, but that this is due to the antagonistic influence on adrenal hypersecretion is still an hypothesis. In schizophrenic disorders, presupposed histological and neurophysiological pathology has remained a theory not supported by facts.

Omitting a discussion of explanations which have been expressed in a phantastic allegorical dress with sublime disregard of present day neurophysiological knowledge, it seems worth while to point to the neurophysiological facts which have been observed and to correlated theories. In the study of respiratory stimulants, an accidental observation was made in 1916 by Loevenhart and Lorenz with intravenous administration of sodium cyanide—after a period of respiratory stimulation, relaxation occurred in catatonic stupor. These experiments were repeated in 1928 with an attempt to get closer to an understanding of catatonic reactions. Upon theoretical grounds, carbon dioxide gas in a mixture with oxygen was used to stimulate respiration, and similar results were obtained. When amytal was used in 1929 to simplify the management of excited patients and to induce sleep in marked insomnia, it was noticed that the state of deep narcosis seemed to change after one to two hours into a natural sleep which lasted several hours and was terminated by a period during which the patient appeared to be clear mentally and in contact with his environment. These observations suggest that anoxemia was an important factor in the results obtained by prolonged sleep and probably also in the insulin treatment. These neurophysiological correlations may point to the fact that it is not a specific drug but the physiological reaction which is essential. It is quite possible that a superior method of producing anoxemia can be found in the near future. It is also quite possible that anoxemia is not the essential factor. In addition it should be kept in mind that the nosological disease entity of schizophrenia has never been established and is much disputed.

A novel development in somatic treatment in psychiatry is the convulsive treatment of schizophrenic disorders, closely bound to recent German genetic psychiatry. It had been well known for over half a century that epileptic patients rarely develop a schizophrenic illness, and that epileptic convulsions are infrequent during a schizophrenic illness. A study of the publications on the occurrence of epileptic and schizophrenic disorders in the same patient is not satisfying. It is often difficult to say whether the patient suffered from a schizophrenic or from a psychopathic condition. Convulsions in schizophrenic patients seem often to have been primarily influenced by affective features. The earlier publications included so-called epilepsy, which was due to arteriosclerotic and

other organic changes which developed in the later life period of hospitalized schizophrenic patients. There are about six cases published in which rapid and unexpected improvement started within a short time after the occurrence of a series of convulsions. A similar observation of a young epileptic patient who was suffering from a deep depression of several months' duration was published by me. Temporary improvement has been described (Dunn [23]) in a 40-year-old patient who suffered from a first depression, when a series of convulsions occurred after amytal administration. These clinical observations demonstrate that the occurrence of convulsions may be followed by improvement in schizophrenic and depressive reactions. Modern genetic studies seem to point to a certain amount of incompatibility between these two illnesses, both of which are, however, poorly defined. The theory that convulsions might produce a biological alteration in the schizophrenic patients and thus lead to improvement seemed to justify the artificial production of convulsions. The results have often been surprisingly good, not only in schizophrenic patients, however, but also in affective psychoses. A perusal of literature shows that in 1781 (Oliver [24]) a manic patient improved after a convulsion caused by administration of a high amount of camphor to induce rest. Less effective results were obtained in the same patient two years later when he suffered from a depression. Although this treatment was noted by psychiatrists, as far as I was able to ascertain there is no record of further repetitions having been undertaken with the definite therapeutic goal of producing convulsions. Minor epileptiform reactions were, however, considered beneficial in the camphor treatment of manic and schizophrenic excitements until about 1820. No satisfactory explanation has thus far been offered. However, it is certainly wrong and most undesirable to call convulsive treatment "specific," especially as the specificity of the illness which it is supposed to affect specifically is unknown.

In any scientific treatment which is not fully understood there is the serious danger of not being able to predict possible damage. It should be an inflexible duty to become thoroughly familiar with the drug or procedure which is to be employed. This implies a thorough understanding of the physiological and psychobiological functions which may have a bearing on the proposed treatment. No one has the right to invent theories to suit his desires without

having given full consideration to what has been established or found plausible. It is a wise and time-saving plan to find out what has been published on the topic, to gain by the success and failure of others. A sufficiently broad historical view will always offer valuable suggestions. Experiments which have been tried when knowledge was more limited may be reviewed fruitfully and theories and methods reformulated. As in the past, there are few attempts to gather all the published material together, and many valuable observations and considerations are lost.

Till near the end of the last century, somatic treatment was honored by the term "medical treatment" and contrasted to "moral treatment" or psychotherapy. There is still an ill-concealed opinion among many psychiatrists that the really valuable treatment will finally be found to be of a somatic nature, and that a physiologically and structurally determined pathology will offer the final explanation of personality disorders. It might be wise to keep in mind at present that the effectiveness of somatic treatment does not prove somatic etiology. It is necessary to consider possible psychobiological factors and study them closely. It is impossible to omit in the consideration of personality disorders the personality involved. Although the psychobiological understanding of emotions has increased greatly, I am not aware that they have been considered sufficiently, if at all, in present day somatic treatment in psychiatry. As in the past, so still today, a detailed and accurate description is rarely carried out in treatment originating in observations which are open to thorough investigation. Much space is given to a description of technique and results, little to a careful description of what can be observed and the patient's experience. There is too strong an enthusiastic drive to help where others have failed or to find an answer to what had been an unsolved riddle.

The pioneer has his place in medical research, and it is desirable to have workers who are not merely making use of the results of others and are not too strongly influenced by the experience of others. On the other hand it is important in medicine to recognize fully the responsibility with regard to those who follow voluntarily, that is physicians; to those who follow blindly, that is lay people; and to those who are forced to follow, that is patients. The pleasure of being an inventor and pathfinder is alluring but leads to all the dangers of adventure.

In conclusion it may be stated that advances in medicine, to which psychiatry belongs, have been dependent more on the general advancement of science than on individuals. Ideas and critical activities are more time-bound than appears to the onlooker at the time of their occurrence. An historical perspective allows us to see interrelations, and new leads may develop. From an historical point of view, it becomes obvious that psychiatric thinking and research have been influenced by two factors: progress and current trends in biology in general, and medicine in particular; and by general cultural developments. Valuable contributions may result from empirical treatment, but it is highly desirable that a scientifically critical attitude serve as guidance. Somatic treatment in psychiatry offers an exceedingly fruitful field, and the well trained investigators should see many possibilities which result from the great advances of recent years in neurophysiology and biochemistry as well as clinical psychiatry.

BIBLIOGRAPHY.

1. Georget, M.: De la folie. Paris, Crevot, 1820.
2. Burrows, George Man: Commentaries on the causes, forms, symptoms, and treatment, moral and medical, of insanity, p. 599. London, Thomas and George Underwood, 1828.
3. Laignel-Lavastine, M., and Vinchon, Jean: Les malades de l'esprit et leur médecine du XVIe au XIXe siecle, p. 267. Paris, Maloine, 1930.
4. Pargeter, William: Observations on maniacal disorders, p. 63. Printed for the author and sold by Smart and Cowslade, Reading; J. Murray, Fleet-Street, London; and J. Fletcher, Oxford, 1792.
5. Haslam, John: Observations on madness and melancholy: including practical remarks on those diseases; together with cases: and an account of the morbid appearances on dissection, p. 315. Second edition, London, Printed for J. Callow, 1809.
6. Pinel, Ph.: Traité médico-philosophique sur l'aliénation mentale ou la manie. Paris, Richard, Caile et Ravier, 1802.
7. Broussais, F. J. V.: De l'irritation et de la folie. Paris, Delaunay, 1828.
8. Ellis, Sir W. C.: A treatise on the nature, symptoms, causes, and treatment of insanity, with practical observations on lunatic asylums. London, Samuel Holdsworth, 1838.
9. Dagonet, H.: Traité élémentaire et pratique des maladies mentales suive de considérations pratiques sur l'administration des asiles d'aliénés. Paris, J. B. Bailliére et Fils, 1862.
10. Griesinger, W.: Die pathologie und therapie der psychischen krankheiten für aerzte und studierende. Third edition, Braunschweig, Friedrich Wreden, 1871.

11. Rush, Benjamin: Medical inquiries and observations, upon the diseases of the mind, p. 181. Philadelphia, Kimber & Richardson, 1812.
12. Brown, J. Crichton: Conium in the treatment of acute mania. Am. J. Insanity, 29: 118-132, July, 1872.
13. Ranney, M. H.: The medical treatment of insanity. Am. J. Insanity, 14: 68, July, 1857.
14. Beard, George M.: A practical treatise on nervous exhaustion (neurasthenia) its symptoms, nature, sequences, treatment, p. 115. New York, William Wood & Company, 1880.
15. Mitchell, S. Weir: Fat and blood: an essay on the treatment of certain forms of neurasthenia and hysteria. Sixth edition, Philadelphia, J. B. Lippincott Company, 1893.
16. Tuke, Samuel: Description of the retreat, an institution near York, for insane persons of the society of friends. Philadelphia, Peirce, 1813.
17. Mesmer, Franz Anton: Mémoire sur la découverte du magnétisme animal. Geneva, 1779.
18. Heinroth, Joh. Christian August: Anweisung für angehende irrenärzte zu richtiger behandlung ihrer kranken. Leipzig, Vogel, 1825.
19. Diethelm, Oskar: Treatment in psychiatry, p. 3. New York, Macmillan, 1936.
20. Carus, C. G.: Vorlesungen über psychologie (gehalten im winter 1829/30 zu Dresden), Zürich, Rotapfel Verlag, 1936.
21. Kläsi, Jakob: Über die therapeutische anwendung der "dauernarkose" mittels somnifens bei schizophrenen. Zeitschrift für die ges. Neurologie und Psychiatrie, 74: 557-592, 1922.
22. Quastel, J. H.: Biochemistry and mental disorder, p. 296. In: Perspectives in biochemistry, edited by Joseph Needham and David E. Green, Cambridge, University Press, 1937.
23. Dunn, William H.: Case presentation. Read before the New York Society for Clinical Psychiatry, New York, 1935.
24. Oliver, William: Account of the effects of camphor in a case of insanity. London Medical Journal, 6: 120-130, 1785.
25. Galt, John M.: The treatment of insanity. New York, Harper & Brothers, 1846. (An excellent review of the treatment from 1689 to 1845 with translations in English from the most important authors.)

DISCUSSION.

DR. D. EWEN CAMERON (Worcester, Mass.).—I think that Dr. Diethelm's final remarks concerning the value of the historical survey method are fully established by the presentation which he has just made. There are certain aspects of this excellent paper which, however, deserve further emphasis.

The first is that one may be tempted to feel that much of the earlier somatic therapy was grotesque and unjustified in its conception. None the less, we must recognize that these attempts to utilize general medical procedures were healthy evidence of a determination to see psychiatry as one of the medical sciences. Indeed it is not hard to imagine that without this bond with the

rapidly developing field of general medicine, psychiatry might have remained for an even longer period under the domination of moral and philosophical considerations. I think, too, that the claims of the lay analyst and of the practicing psychologist would have been easier to vindicate and the whole enterprise of the development of a science of psychobiology would have been greatly retarded.

It is interesting to consider also the underlying conceptions on which these earlier somatic therapists based their ideas. There are perhaps three general contentions which they use to support themselves. The first is that administration of certain agents which act primarily on the lower functional levels such as alcohol and more recently in our own time, benzedrine, undoubtedly produce changes in behavior. The second point is that damage to the organism in the nature of birth injuries or removal of certain of the endocrine glands also produces changes in behavior. These changes are less specific even than those first mentioned because the changes are usually rather a long time in developing and the organism has time to adjust itself individually to the lesion.

The third consideration is that it is impossible to imagine behavior taking place without an underlying matrix. One can't possibly think of a disembodied intelligence, at least scientifically it is impossible to think of that. Consequently it is necessary to imagine that somewhere or other the chain of events which goes to make up behavior must pass through a lower functional level which can be attacked. Both the psychogenic and the somatic forms of approach have been inadequate alone, but of recent years the somatic approach when seen as a part of the general psychosomatic set-up has been considerably more satisfactory.

Dr. Diethelm has mentioned the later developments of Kläsi, and I think it is only fair to point out that Sakel in his presentation of his method has insisted that all possible attention should be paid to the psychic management of the case and the more successful followers of Sakel are those people who have followed that particular line of work.

This new viewpoint is also well exemplified by Lindemann's work with adrenalin and by the work which up to the present has not been recognized, namely, Jacobson's work on muscular relaxation as a means of alleviating certain psychoneurotic conditions.

In conclusion, I think that when the shouting and the tumult in regard to our more recent therapy subside and the captains and the kings who brought these on have departed, we shall find that probably our time will be conspicuous in the historical development of psychiatry as a period during which the first time a real attempt at the development of a psychosomatic psychiatry was attempted and actually achieved.

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THE EFFECT OF TREATMENT ON THE MENTAL LEVEL OF PATIENTS WITH GENERAL PARESIS.*

By SAMUEL H. EPSTEIN, M. D., AND HARRY C. SOLOMON, M. D.,
Boston, Mass.

It has been well recognized that an evaluation of the clinical results in the treatment of general paresis is extremely difficult, and depends upon the personal interpretation of the individual making the decision. The usual criterion of improvement has been the patient's ability to return to work. Some patients are capable of working at the time of beginning treatment, and in such instances the return to work is not to be considered as an index of the success of treatment. In other cases in which disability is a definite feature of the disease, the return to work is dependent upon the economic cycle. At the present time, and for the past several years, many patients who are restored to working capacity following treatment are unable to obtain jobs; and some of them remain in hospital because of the economic condition of the country or of the inability of their families to care for them at home. Furthermore the personal equation of the evaluator plays a large part in determining whether the patient is slightly improved or greatly improved.

Because of these facts it appeared to us to be important to try to get another check on the results of treatment. For this purpose, psychometric determinations seemed to offer a valid method. The advantages are obvious. A single systematic approach to the problem is afforded by psychometry. The method is standard, and the results can be expressed in numerical form. Repetitions of examination afford an excellent check, and above all, since we recognize that a major feature of the paretic process is intellectual deterioration, the intelligence quotient of a patient would seem to be reliable measure of any changes. There is another factor that enters into

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Mrs. A. Louise Collins rendered valuable assistance to this study in analyzing the figures and in the construction of the graphs.

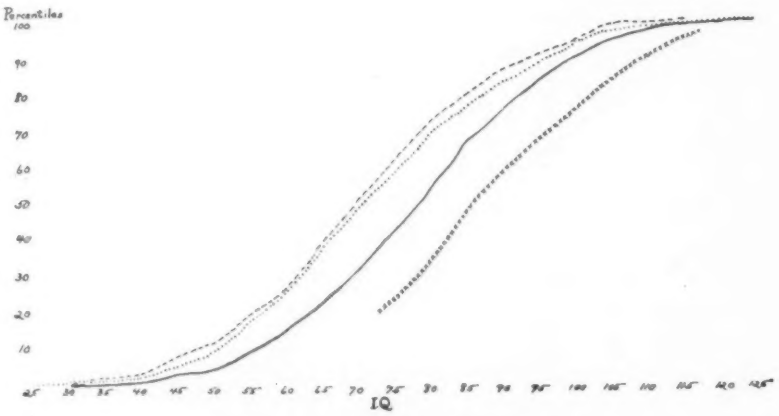
the final clinical evaluation of results; namely, the personality reactions of the patient. However, the repeated psychometric determinations afford at least a standard method of comparing the patient's mental condition before and after treatment.

For a number of years we have tried to obtain the determinations of the intelligence quotient of patients before treatment and at intervals for several years thereafter. This report deals with 78 patients suffering from general paresis who have been treated and who have had two or more psychometric tests, in each case one before treatment and other tests subsequent thereto. In the majority of cases these tests have been repeated over a period of several years. As is true in all attempts to make studies of paretic patients who have undergone treatment, a certain inevitable selection takes place. This is true in the present study because we have chosen for repeated examination patients who have kept in close contact with the clinic and whom we have been able to examine both clinically and serologically. Certain patients were excluded from this study because they were so disturbed mentally that tests could not be made prior to treatment. Other patients died, and still others did so poorly that they were transferred to state institutions and were not subsequently followed by psychometric tests. However the material here presented is, we believe, quite valid in that we are discussing only the results that are shown in these particular cases.

The methods of treatment have varied in this group of cases, in which 57 patients had received fever in addition to chemotherapy and 21 had chemotherapy alone. Our primary interest is not a discussion of which type of treatment gives the best results, but rather what results are obtained as measured psychometrically and how these compare with the clinical estimation of results.

For purposes of orientation we first present graph 1 which shows the distribution of intelligence quotients of four groups of individuals. This is furnished by Mr. C. R. Atwell of the psychological department of this hospital. The solid curve gives the range of psychometric determinations in a large group of patients admitted to the hospital, regardless of diagnosis. The curve marked by crosses shows the levels obtained by a group of drafted soldiers and officers. The curve formed by dashes represents a group of epileptics. The dotted curve represents the untreated paretic pa-

tients. The graph shows similar distribution of the level of intelligence in all these groups, varying from very low intelligence quotients to relatively high ones. It further shows that the intelligence quotients of paretics, while varying within wide limits, are nevertheless considerably lower than those of a fair sample of the general population, as shown in the curve for the army group, and some-



Courtesy of C. R. Atwell and A. L. Collins.

GRAPH 1.

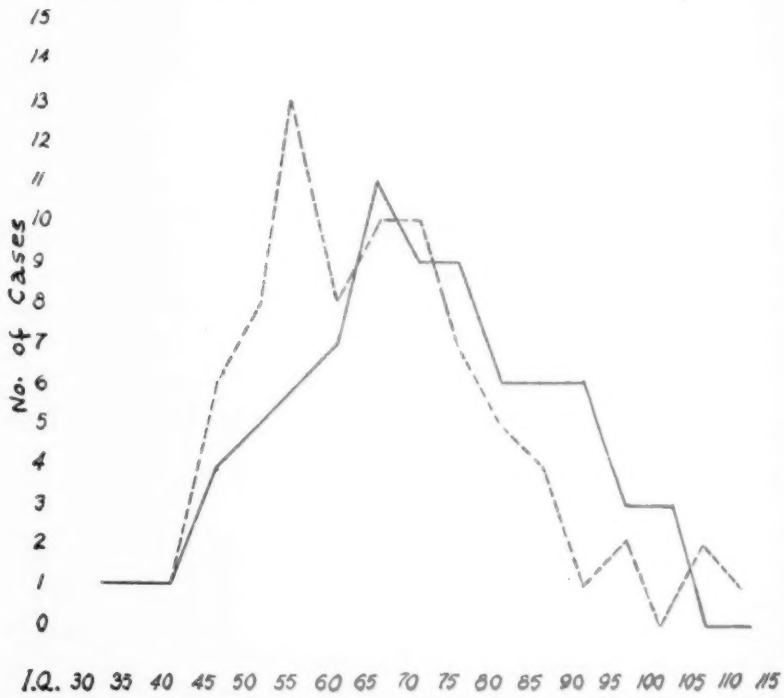
Boston Psychopathic Hospital.
Ogive Curves of I. Q.'s (16 yr. basis).

1,843 Adult Patients (General) ————
229 Epileptic Patients ------
536 General Paretic Patients .....
70,000 (app.) Army Alpha (White Draft and Officers) ××××××

what lower than the psychometric range of general admissions to the hospital.

The distribution curve of this large group of paretics is almost identical in form with the curve of the intelligence quotients of the 78 patients used in this study. Graph 2 presents the distribution curves of the intelligence quotients of these patients before and after treatment. These curves indicate that there is an increase in the intelligence quotients of patients after treatment, taken for the group as a whole. Despite the general increase, a number of patients showed a distinct lowering of their intelligence quotients after treatment.

Graph 3 gives the actual changes in intelligence quotient after treatment, either in the upward or downward direction, and gives the exact material from which Graph 2 is produced. In this graph is shown the original intelligence rating and the amount of change after treatment. Thus the highest dot to the left of the first vertical



GRAPH 2.

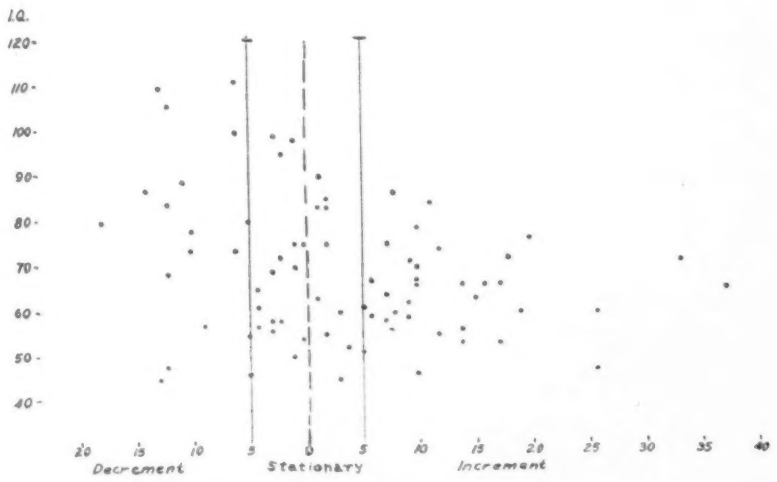
Distribution of I. Q.'s. 78 Patients.

- - - - - - - Before Treatment.
——— After Treatment.

line shows that the patient had an original intelligence quotient of 111, and that after treatment there was a decrease of 6 points, or a final value of 105. Similarly the dot at the extreme right of the graph indicates that the patient had an original intelligence quotient of 65, and that following treatment there was an increase of 37 points, making a final level of 102. From the psychometric stand-

point changes of plus or minus 5 points are to be considered as due to the limitations of the test. Therefore, patients who showed changes of 5 points or less in either direction are considered as having a stationary intelligence quotient.

A study of this comprehensive scatter graph indicates that 31 of the 78 patients showed a change of 5 points or less after treatment, and according to our working hypothesis may be considered as having maintained a stationary level in intelligence quotient. In 10 additional patients there was an increase of less than 10



GRAPH 3.

Relation of I. Q.'s Before Treatment and Amount of Change After Treatment.

points, while 4 more patients showed a similar decrease. Therefore, it is seen that 45 patients showed no change or a relatively small change in the final intelligence quotient. There are, however, certain clear-cut exceptions to this trend where the change is minimal. This is especially marked as regards the rises in the intelligence quotient. There was a rise of 10 points or more in 22 patients, the rise being 37 points in one case and 33 points in another. On the other hand 11 patients showed a fall of 10 points or more, the drop being 17 points in one instance and 14 points in another. There were 7 patients whose intelligence quotient at the beginning was

over 90. None of these showed a distinct rise in intelligence quotient, although in 3 cases the decrease was less than 5 points and in only 2 instances was it over 10 points. From the graph it is seen that the great majority of cases, and the greatest increase in intelligence quotient occurred in those patients having an original quotient of between 60 and 90.

From these facts one may deduce the following conclusions. The majority of patients with general paresis who receive relatively standard treatment for this disorder will have relatively little change in their intelligence quotients following treatment. On the whole, there is a little more likelihood for the intelligence rating to be increased than for it to fall, but this is only a reasonable probability. While the majority of patients will have either a rise or fall of not

TABLE I.

RELATION OF I. Q. CHANGES AND CLINICAL STATUS.

		Increased I. Q.				Decreased I. Q.		
			Increment.				Decrement.	
No. of cases.	Clinical status.	No. of cases.	Aver.	Med.	Stationary. No. of cases.	No. of cases.	Aver.	Med.
37	Improved, working	20	13.6	13.0	11	6	10.0	8.5
24	Improved, not working or hospitalized	8	16.3	13.0	11	5	11.2	10.0
17	Unimproved or dead...	3	11.0	10.0	10	4	12.2	12.0

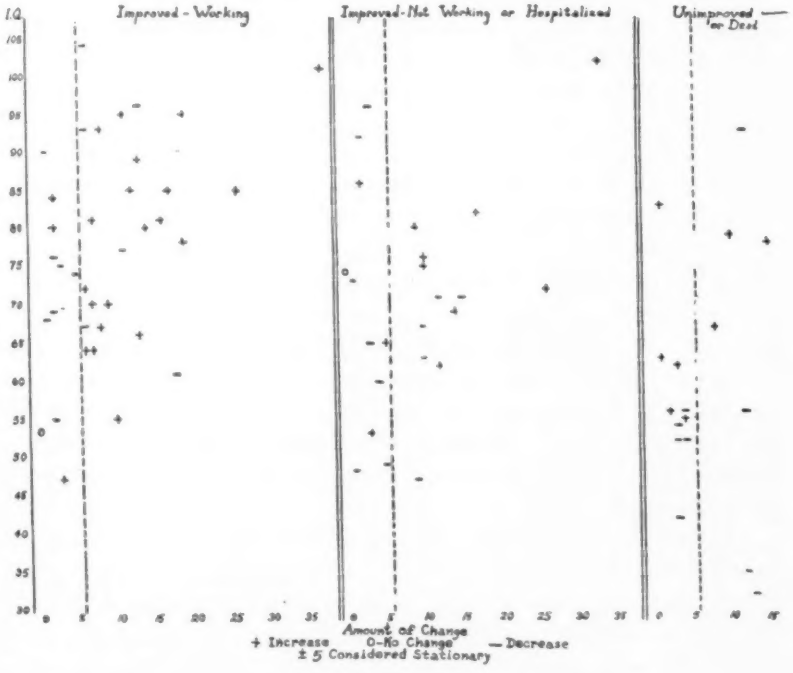
± 5 point change in I. Q. considered stationary.

more than 10 points, there are some very striking exceptions in which the increase or decrease may be very substantial. The patients with an originally relatively high or a very low rating are more likely to show a drop than a rise in their intelligence quotients.

A comparison of the changes in the intelligence levels of the two groups of patients, those treated with fever and those without, showed no striking differences. In view of the relatively small number of cases, one cannot draw too firm a conclusion from this experience. However, there is a suggestion that the results of treatment as measured psychometrically are somewhat better in those patients who had the combination of fever and chemotherapy than those who had chemotherapy alone.

We now turn to a consideration of the comparison of the results as evaluated from a clinical standpoint with the results obtained by psychometric examination, as shown in Table I. In the group of

patients who, from the clinical standpoint, were classified as showing marked improvement with ability to return to occupations similar to their pre-psychotic ones, it is seen that 20 patients showed an increase in their intelligence rating, while 11 patients remained stationary, and 6 showed a decrease which in most instances was slight. Of the 37 patients in this improved working group, 25 had



GRAPH 4.

Relation of I. Q. Changes and Clinical Results.

a final intelligence quotient of 70 or over. This is what one naturally expects, as one would assume that patients with an intelligence quotient under 70 would be unlikely to qualify as efficient workers. However it is found that there were 4 patients in the group who had definitely low intelligence quotients, two with 55, one with 53, and one with 47. It may be well to point out that these four individuals were all foreign born, with language difficulty. The whole clinical material is represented in individual detail in graph 4.

In the second group made up of patients who showed improvement but were not able to return to work or else remained in hospital, the percentage of patients showing an increase in intelligence quotient is smaller than in the first group, and the percentage of those with decreased intelligence quotients is somewhat greater. This fact is more strikingly demonstrated in the group classed as unimproved or dead. It is interesting to note that a few patients in the latter groups had an increase in intelligence quotient, and although they had a relatively high rating, they were nevertheless unable either to work in the community or to be cared for outside of hospital.

One would seem justified in drawing the conclusion that the ability to return to work depends in some degree upon the final level of intelligence as measured by psychometric methods. Moreover the statement may be made that, with rare exceptions, unless the final intelligence quotient reaches at least 70 the ability to be self-supporting will not return. As we have seen from the material presented in graph 3, the change in intelligence of the majority of the cases is likely to be not more than 10 points. One has some reasonable prognostic information from the original pre-treatment intelligence quotients; namely, if the original intelligence rating is much below 70, the chances of getting a patient back to work are relatively small.

Although we do not have material at hand (and it will probably be impossible at any time to obtain the necessary information), it seems that those patients who have had a distinct drop in their intelligence quotients prior to the beginning of treatment are unlikely to make a good recovery, or at least cannot be expected to return to their former occupations. For example, if we take as an illustration a doctor in this group whose intelligence quotient at the end of treatment was 79, we find from the clinical standpoint that this man is economically a total loss, even though he showed an increase of 15 points over the pre-treatment intelligence quotient. While there is evidence of an arrest of progress of the paretic process and no further deterioration after treatment, and although his intelligence rating at the end is considerably higher than that of several patients who have been able to return to their occupations, he is really a dilapidated individual. One has a right to assume that this man who was a successful,

capable practitioner had an intelligence quotient in the neighborhood of 120 before the disease began, so that his final intelligence quotient of 79 probably represents a drop of approximately 40 points, presumably the result of a great deal of cortical cell loss. In other words, we suggest that if the psychometric examination is taken prior to treatment and found to be considerably less than one would expect of an individual as the result of his previous history, education, training and activities in life, one may assume that his chances of making a striking recovery are poor. If, on the other hand, the original psychometric test corresponds with what one would expect, considering the above factors, then one has a right to assume that a satisfying result, as measured by the patient's possibilities of readjustment, is likely.

Delusional and hallucinatory symptoms interfere to a considerable degree with the interpretations arising from the psychometric test. Grandiose delusions are probably an exception to this statement as they are often very fleeting, but delusions of persecution, paranoid formulations and definite auditory hallucinations are less likely to disappear following treatment, particularly if they are well-established. In such cases, the patient does not make a good clinical recovery irrespective of an increase in his psychometric rating, even if the final score may be high enough and near enough his original level to be satisfactory. In other words, such an individual has a personality defect, aside from his psychomotor and intellectual capacity.

There has been considerable divergence of opinion regarding the relationship of the type of psychosis presented by patients and the clinical results following treatment. A consideration, therefore, of the change in psychometric ratings in different clinical syndromes is of some interest. It so happens that of our 78 patients, 33 showed a grandiose euphoric picture, whereas 33 were classified as cases of simple dementia. The remaining 12 patients hardly form a large enough group to discuss. But as we study the 33 cases of the two groups, the following facts are deduced from Table II. The intelligence quotient remained stationary in 15 cases of the euphoric and grandiose group and in 14 of the group with dementia. There was an increase in the intelligence quotient in 12 cases in each group. However, the increment was somewhat greater in the euphoric grandiose patients than in those with dementia, showing

an average increment of 19.3 points with a median of 17.5 in the former, and 11.7 with a median increment of 10 points in the latter. Decrease in the intelligence quotient occurred in 6 cases of the euphoric grandiose group with an average decrement of 12.2 points, while in the dementia group 7 cases showed a decrease with an average amount of 11.2 points while the medians showed a decrement of 11.5 and 12 points respectively.

One may deduce from these findings the general conclusion that there is relatively little variance in results as measured psychometrically in these two groups, with possibly a slight difference in that the euphoric grandiose cases show a little greater increase in the amount of improvement when this does occur. We are inclined

TABLE II.

RELATION OF TYPES OF PSYCHOSIS TO CHANGE IN I. Q.

No. of cases.	Type of psychosis.	Original I. Q. Range.	Original I. Q. Aver.	Original I. Q. Med.	Increased I. Q. No. of cases.	Increased I. Q. Increment. Aver.	Increased I. Q. Increment. Med.	Stationary. No. of cases.	Decreased. No. of cases.	Decreased. Decrement. Aver.	Decreased. Decrement. Med.
33	Euphoric ...	44–109	69	66	12	19.3	17.5	15	6	12.2	11.5
33	Demented ...	45–105	63.6	60.5	12	11.7	10.0	14	7	11.2	12.0
6	Depressed ...	57– 85	71.3	69.5	5	7.6	8.0	1	..	..	..
4	Convulsive ..	83–110	92.8	89	1	11.0	11.0	1	2	6.0	6.0
1	Manic	69	..	..	1	10.0	..	..	..	..	..
1	Schizophrenic.	69	..	..	..	..	..	1	..	..	..

± 5 points change considered stationary.

to believe that this is due in large part to a disturbance of attention at the time the original test was made.

It has long been recognized that the clinical outcome following treatment does not parallel the change in the spinal fluid. Table III shows the relationship of spinal fluid changes to the changes in intelligence quotient. If we compare 32 patients whose spinal fluid became normal with 19 patients whose spinal fluid remained unchanged, it is found that there is no essential contrast in the psychometric results. Thus, in the group in which the spinal fluid became normal, there was just a little less than half of the patients whose intelligence quotients remained stationary, and in the unimproved spinal fluid group, again just under half of the cases showed a stationary intelligence quotient. A slightly higher percentage of the cases developing normal spinal fluid showed an increase in the intelligence quotient when compared with those in

which the fluid remained unchanged, but this difference is not great. Moreover the average increment was greater in the group with unimproved spinal fluid than in those with normal serological results. In other words, the results in the psychometric examination as compared with the spinal fluid change corroborate previous clinical experience showing that there is no essential relationship between the clinical and the serological outcome of patients.

A study of psychometric tests repeated over a period of years shows two important facts. The first is that whatever change occurs in the intelligence quotient occurs rather promptly, that is, in the first months after treatment. The second fact is that after the original change the intelligence quotient in most cases remains

TABLE III.

RELATION OF I. Q. CHANGES AND SPINAL FLUID.

No. of cases.	Spinal fluid.	Increased I. Q.			Stationary. No. of cases.	Decreased I. Q.		
		No. of cases.	Increment. Aver.	Increment. Med.		No. of cases.	Decrement. Aver.	Decrement. Med.
32	Normal	13	16.0	15.0	14	5	12.6	13.0
15	Greatly improved ...	9	10.0	9.0	4	2	11.0	11.0
12	Moderately improved.	5	8.2	8.5	3	4	9.0	9.0
19	Unimproved	6	18.7	5.5	9	4	11.0	11.0

± 5 point change considered stationary.

essentially the same whether there was an original increase or decrease. For example, one patient whose original intelligence quotient before treatment was 72.5, was found two months after treatment to have a quotient of 102.5 and at the end of almost four years the intelligence rating was 102. Another patient, whose original intelligence quotient was 54, after more than four years had a rating of 50. Comprehensive graphs of the whole series of cases appeared so complicating on account of the number of lines that they are not reproduced here. It is interesting to note that the tendency for a change in intelligence quotient to occur promptly after treatment and remain at this level more or less indefinitely, bears no relationship to the condition of the spinal fluid.

SUMMARY.

1. Analysis of the psychometric determinations in 78 cases of general paresis shows that the change in intelligence quotient fol-

lowing treatment is relatively small in the majority of the cases, but there is a little greater likelihood for the intelligence rating to be increased than for it to fall.

2. The greatest increase in intelligence quotient occurred in those patients having an original quotient of between 60 and 90, and the great majority of the cases fell within this range. Patients with an originally relatively high or a very low rating are more likely to show a drop than a rise in the intelligence quotient following treatment.

3. There is suggestive evidence that the psychometric results are better in those patients who had, in the course of their treatment, fever combined with chemotherapy than those who had chemotherapy alone.

4. A comparison of the general clinical results with the psychometric results shows that the ability to return to work depends, in some degree, upon the final level of intelligence; and unless the final intelligence quotient reaches at least 70, the ability to be self-supporting is unlikely. It is also evident that if the original pre-treatment intelligence quotient is much below 70, the likelihood of restoring a patient to working efficiency is relatively slight.

5. It is probable that those patients who have a distinct decrease in their intelligence quotient prior to the beginning of treatment are unlikely to make a good clinical recovery.

6. Delusional trends, if they are well established, interfere with the clinical result even if the final intelligence quotient is high enough and near enough to the original level to presage a satisfactory result.

7. There is relatively little variance in psychometric results in the various mental pictures of the patients in this study, but the euphoric grandiose patients showed a little greater increase in the psychometric rating when this did occur.

8. The psychometric results do not parallel the changes in the spinal fluid, thus corroborating the well established experience that clinical outcome and spinal fluid results show no correlation.

9. If a change in the psychometric rating occurs, it becomes manifest within a few months after treatment, and the level remains relatively stable thereafter.

BILATERAL FRACTURE OF THE FEMORAL NECKS CAUSED BY METRAZOL CONVULSIONS.*

(Report of Case.)

By DONALD C. SOMERS, M. D., and ROBERT P. RICHARDSON, M. D.,
Eloise, Mich.

Despite the severity of the clonic spasms produced by the injection of metrazol in the treatment of schizophrenia, injuries of a severe nature have not as yet been reported in the literature, although many authors have reported injuries consisting chiefly of dislocations of the shoulder and the temporal mandibular joint. The records of metrazol therapy at Eloise Hospital show that 890 injections have been given and 673 convulsions obtained, during which the following four injuries of more than minor character occurred: (a) bilateral fracture of the femur; (b) anterior dislocation of the shoulder with fracture of the greater tuberosity; (c) anterior dislocation of the right shoulder; (d) dislocated jaw.

The reasons for reporting this case are threefold. First, it serves as a warning that, although the percentage of injuries occurring as a result of metrazol convulsions in the shock therapy of schizophrenics is small, some danger does exist and the drug should be used with caution. Second, the method of internal fixation by means of Smith-Peterson nails proved to be highly satisfactory, both from the standpoint of post-operative nursing care and from the standpoint of the end result obtained. Third, no previous report of bilateral fractures of the necks of the femora as a result of convulsions of any nature could be found in the English literature.

The injections are given with the patient lying on an ordinary low hospital bed and no restraint of any kind is used. It is our opinion that restraint would result in far more serious injury than has been the case in this series.

* From the Wm. J. Seymour Hospital, Eloise, Mich.

REPORT OF CASE.

C. N., a white male, age 31, was admitted to the Wm. J. Seymour Hospital May 21, 1938, from the Eloise Mental Hospital with a diagnosis of bilateral hip fracture. Since no history was obtainable from the patient who was a catatonic and unresponsive schizophrenic, it was necessary to obtain a statement from the referring psychiatrist who stated that the patient had been given an intravenous injection of 7 c. c. of metrazol, the ninth in a series of graduated injections of which only one had previously produced a convulsion. The convulsions began with slight twitchings, gradually becoming generalized. They were of unusual severity, and were both clonic and tonic in character. During the three minutes the seizures lasted, one sharp cracking sound was heard. Following the treatment the patient did not move his legs and appeared to be in some pain.

The physical examination revealed a fairly well nourished, pale young man of tall stature, lying quietly in bed and apparently not in acute distress. He was very apathetic except for repeated grimacing, and showed a complete lack of interest and response. The lower extremities were externally rotated and slightly adducted. There was some muscle spasm present. Other clinical and laboratory findings were as follows: blood pressure, 118/82; rectal temperature, 100.4° F.; pulse, 76; respiration, 20; urine: sp. gr. 1.023; albumin and sugar negative; occasional white blood cells. The blood picture was: hemoglobin, 12.5 grams; red blood cells, 3.9 million; white blood cells, 9,450; Kahn, negative. Radiographic studies revealed fractures of the femoral necks (Fig. 1).

TREATMENT.

Temporary skin traction was applied, using 10 pounds of weight bilaterally, pending permission for open reduction. On June 4, 1938, under general anesthesia, each femur was reduced separately and placed in moderate abduction, extension and internal rotation. Through a four-inch incision over the greater trochanters, fixation was accomplished with Smith-Peterson nails. Radiographs taken during the operative procedure revealed that reduction and fixation had been obtained.

The patient had an uneventful post-operative course. Skin traction of 10 pounds, bilaterally, was used for six days following the operation. He was allowed up in a wheelchair on the eighth post-operative day, and has continued to be up twice each day since that time for as long as he wished. It was not considered advisable for him to walk because of his mental incompetence, which prevented him from using due care. However, on August 3, 1938, he did walk without permission and apparently no harm resulted. The only special care this patient was given during his convalescence was passive flexion of the knees and hips twice daily for eight days, active and passive motion twice daily for the following week, and unlimited active motion since that time. Since no casts were used, the usual anxiety about complications such as pneumonia, decubitus ulcers and stiffening of joints, was obviated. The patient has been walking for approximately two weeks

FIG. 1.—Demonstrating Bilateral Intracapsular Fractures.

FIG. 2.—Demonstrating Degree of Union Present Fourteen Weeks Subsequent to Operation.

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and his present condition is excellent. Check radiographs 14 weeks following reduction reveal solid union in the right fracture, and sufficiently solid union on the left to permit weight bearing (Fig. 2).

COMMENT.

This paper is not presented as an argument against the use of metrazol, rather the record of injuries sustained is so favorable as to encourage its use. Nevertheless, the fact that through muscular contraction alone the necks of normal femora were fractured in a 31-year-old man, indicates the potency of metrazol and the care with which it should be employed. Its use is certainly contraindicated in any condition where bony atrophy is present, due either to prolonged recumbency in bed or to systemic disease.

THEORY AND PRINCIPLES OF THE "TOTAL PUSH" METHOD IN THE TREATMENT OF CHRONIC SCHIZOPHRENIA.*

By ABRAHAM MYERSON, M. D., Boston.

This paper presents an amplification and synthesis of well-known methods of approach to the treatment of the chronic schizophrenic. The only claim to originality lies in the general underlying theory and in the aggregation of forces used, this being the basis for the term "total push." The term total push is graphic rather than exact, since nothing can be total, and the forces used do not necessarily conform to the crude word push but only to its meaning of power or force.†

Theoretic Basis.

It is here claimed that hospitalization of a patient under the circumstances of the care usually given produces a "prison stupor" or a prison psychosis which interacts with the social retreat of the original schizophrenia. Schizophrenia is, in certain of its characteristic manifestations, a retreat from social contact into delusion.

* Read before the Massachusetts Psychiatric Society January 27, 1939.

† Some psychiatrists who have heard this paper read have stated that it is reminiscent of the Bernard Aschner treatment of chronic schizophrenia. There are important differences. In his treatment the emphasis was laid on drastic physiologic and psychologic means, such as catharsis, apparatus for shaking about the patient, hormones and especially as applied to his material which was female mainly. This is quite opposed to the treatment here outlined, in that no drastic and non-physiologic means were used, nor was fear of any overt type instilled in the patients. The essence in the treatment is an increased humanity in the approach to the problems of these patients. Furthermore, he claimed a cure for schizophrenia which is not at all claimed in this paper and for this method.

The leading principle is, of course, a continuation in the direct line of all the efforts of psychiatry and psychiatrists for the past hundred years and more to bring an enlightened pressure to bear upon the mentally sick patients. What is here claimed is that a thorough-going steadfast pressure of humane and physiologically sound background has been utilized in a complete sort of way.

The negativism and the cerea flexibilitas of the pronounced catatonic type represent the extreme form of this retreat. The history is that of a gradual or sudden withdrawal from ordinary social relationships into solitariness, lowered initiative, general passivity, and with this are often associated physiologic disturbances in the functions of the organism, such as impaired sleep, vasomotor reactions usually of a passive type, and lowered capacity for exertion. The individual finds social relationships and contacts either linked up with delusion, or painful in some way or other. There is commonly the feeling of reference, of unfriendly attitude on the part of others, and inability or unwillingness to face and to hold converse with others.

Such a patient is put in an institution. Immediately whatever remaining initiative he had is taken from him. His social contacts become even more diminished. He becomes entirely passive in his activities. He is immersed in monotony and perhaps, most importantly, he lives in a *motivation vacuum*. That is, reward and punishment disappear from his life. He is neither praised nor blamed for his conduct. The ordinary man depends on these motivations for the bulk of his conduct. The fear of the law and, more potently, the fear of the ill-opinion of others operate with enormous force in the lives of everybody, and together with this, praise and reward act as tonics to conduct which never lose their potency. In other words, the psychologic retreat of the schizophrenic is enhanced in all directions by the usual hospital care which he receives.

A physiologic vacuum also operates. With the best will in the world the institutions do not provide adequate exercise, adequate out-of-doors and sunshine, and unless the patient shows a willingness to work, there is neither the time nor the inclination to push such a patient into activity. So on the back wards of all hospitals, one finds the chronic schizophrenic, who sits on benches, stands in a corner or paces automatically to and fro, grimacing, passive and absorbed in his delusions, bursting forth now and then into spasms of hostility and aggression and then subsiding into his automatisms.

This retreat is not so deep as it seems, and I will state at this point that from all the evidence we have at the present time the symptoms of dementia præcox are not so deeply grounded nor so unalterably related to lesion as we have assumed. Even the fact of

its heredity does not necessarily involve hopelessness, since we have the paradigm of diabetes to encourage us in the treatment of the constitutional diseases.

The above is demonstrated, first, by the spontaneous recovery which continually takes place, and the return from apparent marked deterioration to a condition much better and immensely surprising. Second, the results with such drugs as amytal or alcohol indicate that for a time, at least, the individual returns from his retreat to something much more nearly normal. Following amytal, the patient may talk very coherently and with a great show of animation, may interchange social remarks in a fairly adequate manner for a short time. As the amytal wears off, the patient slips back into a shell of retreat and delusion. The shock treatments, whether by metrazol or insulin, indicate that the dynamics of the disease can be altered by agents which operate both physiologically and psychologically. We need not be discouraged by the fact that these drugs have not lived up to their early promise. The airplane has flown ten yards; it has crashed; but the fact remains, it has flown ten yards.

We may, therefore, assume that whatever lesion may exist is not extensive; that what we deal with here may be mostly a personality imbalance, very much like the physiological imbalances produced by agents which create over-activity of one group of functions, such as those sympathetically induced and which produce imbalance between sympathetic and parasympathetic activity. The organism is an ecology—chemical, physical, psychological. Disturb the ecology in one place, and one gets a change in activity of the organism which is surprisingly diffuse and may involve functions apparently far away, so to speak, from the new factor introduced.

Partial Push.

Since the days of Pinel, the attempt has been made spasmodically and partially to substitute a more human atmosphere for that of the prison hospital. Hydrotherapy, physiotherapy, the use of drugs, diet and exercise, as well as occupation and entertainment, have played a rôle. These have not, however, been used with completeness or thoroughness.

An experiment in this general direction has been carried on at the Boston State Hospital for the past two years. Every patient, who is

not bed-ridden, has been removed from the ward at least once a day, weather permitting, spring, summer, autumn and winter. Far more hydrotherapy has been used than previously. The diet has been amplified. Some degree of exercise, though not in great measure, has been brought into the care and handling of the patients at the hospital. Entertainment has been enormously increased. For every day of the year there is some entertainment. Local groups of entertainers have volunteered their services, and the general life of the patient has thus been altered in the direction of more sunshine, exercise, hydrotherapy, feeding and entertainment.

The result has been a definite improvement. The number of deaths has definitely diminished. The number of discharges has increased. The necessity for restraint and sedation has been greatly diminished. In tangible and intangible ways the mental and physical health of the patients has improved in this widespread approximation to more normal living.

Similar experiments have been carried out for a short period of time at the Grafton State Hospital. The results have not been tabulated at this institution, but in general I am informed there has been improvement.

Total Push.

With this partial push as a basis and on the theory that the prison stupor or psychosis played a rôle in the increasing retreat of definitely chronic schizophrenic patients in hospitals, I evolved the method of "total push." The term "total," I repeat, is misleading in a certain sense, since totality is infinity and this cannot be approached. Moreover, the various forms of the push used are introduced into the life of the patient in a sort of series and gradually made more operative. However, a technique has been evolved which merits the term "total" as a relative designation and has the value of arousing interest and cooperation. This technique is based on the introduction of the following measures:

I. GENERAL MEDICAL MEASURES.

1. Physiotherapy.—It is probable that the first and easiest approach to the chronic schizophrenic is by the utilization of vigorous physiotherapy. The value of showers, douches, massage and rub-

downs to give, first, a start to the patient's energies, and second, because of their general tonic effect on the organism, has been demonstrated by our experience. Therefore, there should be the utilization of these measures in a routine, steady fashion, day by day.

2. *Irradiation.*—We especially stress the use of ultraviolet irradiation on the basis of some experimental work done by Dr. R. Neustadt and myself, in which it has been shown that the sex hormones excreted in urine increase quite markedly following ultraviolet irradiation, especially of the testes. This fact, while not related insofar as we know at present to the general well-being of the individual, seems sufficiently important to incorporate ultraviolet irradiation as a unit of our total push.

Associated with this matter of ultraviolet irradiation is the necessity for out-doors, and this can be incorporated in the form of walks, etc.

II. EXERCISE AND GAMES.

One of the essential difficulties in patients of the type which has been treated is the inertia and even the resistance of the patient to games and exercise. We have found, however, that it is often easy to overcome this, and we have followed to some extent the method discussed by John W. Thompson in his as yet unpublished work on exercise in the treatment of schizophrenia. If the patient will not actively exercise at first, passive motions carried on by a physical trainer break down this resistance very readily. The mere throwing of a bean-bag to women and a medicine ball or ball of other type to men generally succeeds finally in bringing the patient into cooperation in this direction. It can be said without much elaboration that the inertia is readily broken down and some degree of cooperation as readily established. After this breaking down of the original inertia, formal exercises in a gymnasium under the direction of a physical trainer or one qualified to teach is of great value. Our experience has been that in a relatively short time the patients cooperate in games of all kinds, go through exercise with vigor and with considerable skill in a manner quite surprising in view of their general inertia on the wards.

The extension into actual work of these patients has not up to the present been undertaken. This represents, however, a definite later phase of push and will be reported upon at a future time.

The games used have ranged from bean-bag throwing and such games for women to badminton, pool, billiards; competitive running, jumping and formal athletic activities. The patient is pushed or transferred from the less strenuous and more simple activities to those requiring coordination, skill and spontaneous interest as soon as possible.

III. DIET, VITAMINS, ETC.

An increased and more liberal diet, together with eating in common with others, and a general shift from the tray in the back-room to the æstheticism, companionship and sociability of the dining-room, is made as soon as possible. It is often surprising how quickly this can be done and how the patients respond, even in the case of people who have not stirred from their rooms for years. To bring this about, motivation, which will be discussed later, has to be used; but when it is used, it operates well and becomes a desired activity; that is, the patient himself seeks the enhanced æstheticism, sociability, and what may be called the civilization of the situation.

The theory is, of course, that latent æstheticism is aroused and the monotony which seems to be sought by the patient is broken up so that a desire to escape it takes place. What the patient feels in this matter, we have not sought to determine, since up to the present no attempt has been made to discuss the psychology of the situation with the patient.

Vitamins.—In certain patients we have used small doses of insulin to awaken appetite. We have given vitamins in the form of the various combinations, not with any more definite indication than the general appearance of the patient. Vitamin deficiency in an organized form does not occur frequently in the schizophrenic. Nevertheless, we have felt that it might have value and because of the general concept of total push, we have used vitamins to some extent.

We have not, however, used the hormones at all.

IV. PSYCHOLOGIC PUSH.

1. Clothing.—Many of these patients have been destructive of their clothes, untidy, and consequently the care of them in this

respect has lagged to the point where they lived in bathrobes or in garments which satisfied elemental needs but which did not express personality and were not conducive to the awakening of æsthetic feeling or of pride in the personal appearance. To change this has been one of the primary approaches to the problems these patients present. The patients have been dressed up. They have been shaved regularly. Their nails have been cared for. Their hair has been treated, in the case of women. Even if they at first destroyed their clothing, some punishment or deprivation has been introduced, and it may be stated that in a short time, these patients wear good clothes and take reasonably good care of them. There is an enormous difference between the human being dressed properly and the same individual wearing unattractive or disorganized clothing. One of the sources of the human being's egoism is his clothing. One of the principal entries to normal, human social relationships is in what men wear (and particularly in what women wear).

2. *Praise, Blame, Reward, Punishment.*—Praise and blame have not been utilized so much as reward and punishment. Reward in the form of more privileges, candy, ice cream, and the retention of the added food-stuffs, delicacies, cigarettes, cigars—the giving and taking away of these rewards and punishments has constituted the main motives up to the present time. It would be interesting, but entirely impossible, to see what the effect of physical reward and physical pain would be, but society has not yet developed to the point where certain privileges and physical punishment can safely be used.

Motivation, however, does reside in praise and blame. These have been utilized liberally. Praise in the presence of the group and reward under the same circumstances are important. At present we have not reached the point where group therapy has been largely used, although we will do so as the patients develop in sociability. The work in this respect, which has been described by Rhoades, Low, Marsh, Wender, Dunham, and Farris, undoubtedly constitutes an important phase of the total push.

We have not as yet reached the phase of education in the formal sense, except in the case of one patient. This will be described by Dr. Tillotson in his paper. A patient at the Boston State Hospital, who was formerly a good chess and checker player, has been rein-

troduced into these games and already plays an excellent game in both of these fields of intellectual exercises. Cards have also been utilized in his case.

We believe in teaching music, dancing, craftsmanship and general occupational therapy, and these will be utilized as the patients grow, if they do grow, in responsibility and general internal organization.

Results.

Results thus far of the "total push" method have been gratifying and will be discussed in detail by Dr. Tillotson. It is not here assumed, claimed or believed that this is a cure for schizophrenia, the contrary being the assumption made. The experiment thus far shows, however, (1) that the deterioration of the chronic schizophrenic, by which we mean individuals who have been mentally sick in hospitals anywhere from 6 to 20 years, who have never shown any tendency to remission or improvement, who have gradually taken on the aspect of what is called deterioration, is not altogether a natural or an entirely necessary product of the disease; (2) that it can be wiped off at least in part by effort and the patients restored to a more human and active appearance. What the future will show for this method as to its relationship to cure is for the future to determine.

It may be added that a sort of prison stupor is one of the dangers that threatens the hospital attendant and the hospital physician. The introduction of these measures by our present experience is a great tonic to the spirit and energy of the attendants, nurses and doctors, and if it did nothing more than this it would be exceedingly worthwhile.

THE PRACTICE OF THE TOTAL PUSH METHOD IN THE TREATMENT OF CHRONIC SCHIZOPHRENIA.*

By KENNETH J. TILLOTSON, M. D., Waverly, Mass.

The preceding paper, in which Dr. Abraham Myerson has outlined the theories and principles of the "total push method," forms the basis for the following clinical study. The problem may be succinctly stated as follows: Is it possible by concentrated therapy of the types described by him to effect beneficial changes in deteriorated patients suffering from some type or types of schizophrenia?

About November 1, 1938, the total push method of therapy was organized and applied to a group of 11 patients; all of these patients with one exception were suffering from some type or types of chronic schizophrenia. During the past month 22 additional patients have been receiving this same method of therapy. So, at the present time we have 33 patients of the so-called chronic deteriorated types who are being treated by the total push method.

Before reporting upon these patients and the clinical results of this treatment, let me refer to an important paper for the sake of a clearer orientation. Cheney and Drewry published a paper (American Journal of Psychiatry, Vol. 95, No. 1, July, 1938) on "the results of non-specific treatment in dementia præcox at the Bloomingdale Hospital based on a follow-up study of 500 schizophrenia patients" in a private hospital of the McLean type. This article stresses the intensive and individual care and treatment by well established methods. Cheney and Drewry state further that:

> In patients with an average or higher intelligence, remaining for treatment for approximately one year, it may be expected that at the end of their hospital residence 37 per cent of the total number will be benefited by the treatment and 7 per cent will have recovered. If the patients are of the catatonic type of dementia præcox the prospect will be that 42 per cent of them will be benefited, with 10 per cent recovered. The prospect of improvement in the paranoid, hebephrenic and simple types will be less, but for patients suffering from other forms than those already mentioned it may be expected that more than half will be benefited by treatment and 17 per cent will be completely recovered.

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* Read before the Massachusetts Psychiatric Society, January 27, 1939.

If the patients have shown symptoms for not more than six months before admission, it may be expected that half of them will be benefited and 10 per cent of these will be recovered. If the illness has existed longer there will be less expectation for improvement and recovery but the latter may occur even if the symptoms have existed for several years before admission.

If the dementia præcox patient remains under treatment for six months to a year he has a better chance of improvement than if he left before six months' residence. If he is not improved by the end of one year, there is less chance that he may recover even after two years' treatment in the hospital.

Furthermore based on the study we may expect that from 2 to 12 years after admission of these 500 patients, 10 per cent will have died, *43* per cent will be having continued care in mental hospitals, and the remainder will be living outside such hospitals. Approximately 60 per cent of the original group will be unimproved, 16 per cent will be improved, 14 per cent will be much improved, 12 per cent recovered completely.

This aids us in orientation because it is the 43 per cent of chronic patients remaining in the hospital with whom we are chiefly concerned at the moment. What can be done with this 43 per cent? It was hoped that the shock therapies might prove beneficial in a considerable number and their application has been studied with interest. They have had some beneficial results although in most instances finally disappointing. These patients who have been treated with insulin and metrazol have been referred to as patients receiving a "specific" form of therapy. The foregoing report of 500 cases of dementia præcox treated in the Bloomingdale Hospital and the results were presented by Cheney and Drewry to offer a comparison of the results of treatment of an intensive individual type as compared with the specific method of therapy. It is not the purpose of this paper to make any comparisons between total push treatment and shock treatment or other forms of therapy.

With this program in mind we are now studying our patients before starting them on the total push method as though they were newly admitted patients. In order to evaluate the changes which we may now reasonably expect to take place, we suggest the following work-up at the beginning of the total push treatment. A physical examination including urinalysis and blood chemistry should be done. An important examination from our point of study is the electroencephalogram. A mental status and a psychometric and Rorschach test if possible should be done. Most of these tests and studies have been attempted and completed to date in many of the

33 patients we are treating with the total push method. This method seems to be a logically progressive procedure for the reintegration of these patients. To determine the full significance of the following changes the foregoing detailed studies seem necessary.

CASE REPORTS.

PATIENT 1 is a male, 28 years of age. His education consisted of six months in college and he had no occupation. He had been ill with schizophrenia of the catatonic type for four years and his total hospital residence at the beginning of the treatment was three and one-half years. A year ago he was given six and one-half months of insulin therapy combined with metrazol; his maximum dosage of insulin was 180 units. After this treatment he showed improvement but this was followed by relapse. Before beginning the total push treatment this patient was seclusive and uncooperative; attempts to obtain cooperation and routine threw him into rages. He refused to cooperate with any program and was cared for on a disturbed hall.

The total push method was applied and a quick response was noted, especially on the reward-punishment basis; he was denied cigarettes unless he accomplished certain acts and cooperated in his routine. He was rather forcibly coerced into activities and into his physical program of exercise in the gymnasium and walks. Games of badminton, bowling, pool and billiards were gradually developed. The patient was transferred to a better hall and his range of activity has developed to the point where he spends six hours a day in active games and walks. He goes to the physiotherapy department where he receives medical baths and ultra-violet radiations. He is taking greater interest in his personal appearance and is more friendly and sociable. His skill in games has improved; there is a slight lessening of his mannerisms and he is less negativistic and less preoccupied, maintaining in general much better contact with reality. There has been a slight improvement in his physical status.

PATIENT 2 is a male, age 17, a grammar school student. He has been ill with schizophrenia, other types, for five plus years and has been in three different psychiatric hospitals. A year ago he was given seven months of insulin treatment combined with metrazol; the maximum dosage of insulin was 100 units. He improved somewhat under this form of therapy but had definitely started to regress and his condition was that of rather typical hebephrenia.

Following the total push program his activities were markedly increased. He attends the gymnasium where he takes active exercise and plays games, such as badminton, bowling, pool and billiards, in an interested and efficient manner. The reward-punishment theory has worked very well with him; he enjoys playing card games which he is allowed to do as a reward. He has been able to go for week-ends with his parents and a nurse; more recently he has been able to go home without a nurse and has made a normal adjust-

ment in his home environment. His æstheticism is manifest in his interest in music and art and in his personal appearance. His mood and sociability are improved; he is more friendly and his contact with nurses, physicians and fellow patients here is markedly changed for the better. He plays games better and with an active interest. There is little change in his mannerisms which were not marked except for his manner of speech. His contact with reality and general interest are improved. During the recent weeks this patient has made such marked improvement in his social reactions and in his conduct that a re-educational program at the grammar school level has been resumed. This was done after a comparative check of his I. Q. and Rorschach test with similar tests that were done in 1935. His I. Q. in 1935 was 133; at present it is 101. The Rorschach had not changed materially. It is interesting to note that this patient was concerned with his I. Q. rating. He is now taking grammar school arithmetic and English and piano lessons and is making fairly satisfactory progress in the latter. He has ultra-violet treatments and is on a high vitamin diet.

PATIENT 3 is a male, age 38, a graduate of the U. S. Naval Academy; at the onset of his illness he was a naval officer on duty. The patient has been ill 12 years and has been in a hospital over nine years with a diagnosis of schizophrenia, hebephrenic type. Before the total push method began this patient was uncooperative and was cared for on a disturbed hall. He was irritable and used profane and vulgar language. He was disheveled in his personal appearance and refused to cooperate. Occasionally he went out for walks in the yard adjacent to the building. He refused to go to the gymnasium or the moving pictures and would not participate in any form of diversion, entertainment or occupational interest.

Since the total push he has developed an active program of games and exercise in the gymnasium—calisthenics, badminton, bowling, pool and billiards—and he takes interest in showing his skill in these games. He has become interested in his personal appearance and has had his room changed to a quiet hall. He now greets physicians and nurses in a friendly manner, is quite responsive in a gentlemanly way, frequently smiles, at times shows aggressiveness in a bluffing manner, is less preoccupied and seems to have full and active contact with reality. He likewise is receiving medical baths and ultra-violet treatments and high vitamin diet. He devotes seven hours a day to his program. His mood and sociability have markedly improved. He plays cards with his fellow patients and in games with the other patients and nurses he is friendly. His original athletic ability is manifesting itself; he plays games fairly skillfully. There is a marked lessening of his dilapidated appearance; his contact with reality and his general interest are much improved. His physical status has likewise improved.

PATIENT 4 is a male, age 50, a college graduate and bond salesman. He has been ill 25 years and has been in the hospital continuously with a diagnosis of schizophrenia, paranoid type. Before treatment he spent practically all his time lying on his bed although he was dressed and went to the dining

room for his meals. He was secluded in his room or paced the corridors cursing under his breath. Occasionally he went outdoors into the garden but was uncooperative in any attempts to interest him in games; he was exceedingly vulgar and profane when such efforts were made. Earlier in his illness he was extremely aggressive and homicidal; this extreme aggressiveness was not shown in physical violence in recent years but his verbal abuse continued.

Since the total push he has developed a very active program. He goes to the gymnasium twice daily where he first was engaged in calisthenics and exercises and reluctantly and protestingly yielded in cooperation. He not only does these exercises but he plays badminton, bowls, plays pool and billiards and is interested in card games with other patients. Likewise, he has the medical baths and ultra-violet treatments. He frequently asks other patients and nurses to join him in games. He has taken a great interest in his personal appearance; originally he was fastidious in his dressing habits and this interest in dress has returned. His program, therefore, has markedly improved as well as his contact with reality. He spontaneously attends to his toilet needs and even brushes his teeth regularly. He has cooperated fully with the active program of a convalescent patient.

PATIENT 5 is a male, 43 years of age, a grammar school graduate and who was working as a draftsman. He has been ill and continuously in the hospital for the past 19 years. His diagnosis is schizophrenia, hebephrenic type. Before the total push this patient was extremely negativistic, refusing to leave the corridors except to go occasionally for walks in the yard adjacent to the building. He refused to attend the occupational center and when he did he became so disturbed that it was necessary for him to be returned to his room. He frequently assaulted other patients and nurses. He showed no interest in any form of occupational diversion; in fact, his status was characterized as apathy with periodic outbursts.

Following the total push treatment this patient developed a program rather promptly upon active urging and pushing. At first he resisted and protested. Nevertheless he went to the gymnasium where exercises, medicine ball tossing and ring toss were instituted. As in all these cases he was given stimulating medical baths and ultra-violet treatments. He now attends the gymnasium twice a day. He plays games of various types, particularly badminton, pool and billiards and does some bowling, and he maintains an interest in these games in which he shows a moderate skill. He has recently become interested in clay modelling. He now responds very well to praise and is sensitive to criticism or blame. He is taking much better interest in his personal appearance and has started to play the piano; he was formerly interested in music. This development of activity, and the improvement in his æstheticism, mood and conduct with a lessening of his mannerisms are marked.

PATIENT 6 is a male, age 44, a graduate of college and had one year in law school. He was a stock and bond salesman at the onset of his illness. He has been ill over eight years suffering from schizophrenia, paranoid type.

One year ago he was given seven months of insulin therapy combined with metrazol; the maximum dosage of insulin was 125 units. This patient did not respond to the insulin therapy, in fact his condition following this therapy was recorded as slightly worse. Before the total push treatment this patient had withdrawn from his activities and was suspicious, uncooperative and expressed marked delusions of a paranoid type.

Under the total push he has resumed, with only a partial degree of cooperation, a program embracing the activities of the foregoing patients; that is, he goes to the gymnasium twice a day where he is more or less pushed to do calisthenics and games which he plays fairly well except that his interest at times lags and he becomes preoccupied frequently in the midst of a game, having to be reminded that he is playing a game. His interest, therefore, is not maintained but his scope of activities and the types of games he plays—badminton, bowling, pool and billiards—has improved. His mood is less irritable. He has disliked very much the hydrotherapy treatments and the ultra-violet radiations. The reward-punishment principle has been utilized in his case by trying to improve his appetite by discontinuing his baths and having him continue as far as possible with the games and physical program. This patient (by virtue of total push) is maintaining his program.

PATIENT 7 is a female, age 62, who has been ill for a little over two years. This patient had two years of college work and she has never had any occupation. She is a maiden lady whose personality throughout the years before the onset of her acute psychosis which necessitated hospital care two years ago was of such a nature that it seemed proper to include her in this group since like all these patients she was considered a chronic, deteriorated, hopeless type. Before the total push method was started she was combative, liable to attack others, rarely occupied and showed little interest in her surroundings.

Since the total push treatment began she has developed a fairly active program. She attends a gymnasium and occupational therapy department, takes walks, is interested in puzzles, chats with the other patients and with the nurses and physicians in a more friendly manner. She attends the movies, teas and other social activities in the hospital, having therefore much more contact with reality. Her mental picture of agitation and extreme depression has improved and her somatic delusions have disappeared and are never referred to. Her physical condition is markedly improved. She is having the hydrotherapy baths, vitamins and ultra-violet treatments and is taking an active interest in her personal appearance. She goes to the beauty parlor for treatments and there has been a marked improvement in her physical status.

PATIENT 8 is a female, age 41, a grammar school graduate and was taking a course in practical nursing. She has been here in the hospital continuously for the past 18 years with a diagnosis of schizophrenia of the hebephrenic type. Before the total push began this patient was considered completely deteriorated and demented and because of her destructive tendencies and excitement spent seven hours daily in the continuous baths. At other times she was in seclusion where she was disturbed and untidy, rarely depressed.

Since the total push method began she has been on a modified program; the modification is that she is rarely in the baths for more than three hours daily. She goes to the gymnasium where she plays badminton, ping-pong and other simpler games. She does clay modelling in the occupational department. On the hall she is dressed, conducts herself in a socially acceptable manner and plays simple music on the piano. She therefore shows a marked improvement in her contact with reality and shows a certain amount of industry, skill and æstheticism. There is improvement in her mood and in her general behavior and conduct.

PATIENT 9 is a female, age 41, married, housewife. She is a graduate of private school and had some high school work. She has been ill 12 years and has been in the hospital about five years with a diagnosis of schizophrenia, catatonic type. About a year ago she had a metrazol therapy, 10 doses over a period of about three weeks, followed by a few lucid intervals and temporary improvement. Her condition before the total push therapy began was one of extreme aggressiveness with homicidal tendencies, and she was in a continuously disturbed state. She spent four hours daily in packs and the rest of the time in seclusion. She was unapproachable for the foregoing reasons.

Since the total push began this patient has been out of her room part of nearly every day. She goes to the gymnasium about three times a week where she plays ping-pong, basket ball and other simpler games. She is dressed daily and about the ward and goes to the garden for long walks. She has shown a marked improvement in her mood and social reaction. She is more friendly. At times packs are combined with this program instead of the other types of hydrotherapy. Her sleep has improved. Her contact with reality and her general interest have improved. She is making a much better adjustment in her conduct, is less aggressive and at times, for brief periods of an hour or two, makes an acceptable social adjustment.

PATIENT 10 is a female, age 43, housewife, who graduated from private school and had three years in college. She has been ill for over 11 years, has been in several hospitals and is diagnosed as schizophrenia, paranoid type. Before the total push treatment began she spent most of her time in seclusion, was disturbed and combative.

She is now on a partial program which is gradually being increased. She is in seclusion very little and her disturbed periods are less marked. She has developed an interest in piano playing and attends the gymnasium three or four times a week where she plays badminton and she goes for long walks in the garden. Her æstheticism has markedly increased; she is interested in the use of cosmetics and facial creams which are given her as a reward following out the reward-punishment principle. She takes her meals in the dining room. She shows improvement in her mood and sociability and in her general contact with reality. Her program of interests is considerably broadened.

PATIENT 11 is a female, age 47. She was a housewife whose education consisted of private school and finishing school. She has been ill for 19 years and has been in the hospital five years with the diagnosis of schizophrenia of the hebephrenic type. Before this program began the patient remained in her room most of the time and when out of her room was preoccupied, staring and apathetic. She was resistive and untidy and very much inhibited and blocked by her extreme mannerisms.

Since this total push program was instituted she has developed some interest in activities; she attends the occupational therapy department where she does fancy work and she does pottery or clay modelling. She enjoys moving pictures and entertainments and tea parties. She plays the piano and sings songs in a low voice while on the hall. She is much more sociable, less inhibited and actively cooperates with the foregoing program. She is interested in her personal appearance and in music; to this extent her æstheticism has improved. Instead of being irritable she is now amiable and greets visitors in an appropriate manner although she is rather shy and diffident in her manner. Her contact with reality and her general interest have improved. There is improvement in her physical status.

SUMMARY.

The foregoing group of 11 deteriorated patients, suffering from chronic schizophrenia, who have been ill an average of 12.1 years and whose average hospitalization is over ten years, have shown the changes as recorded after three months' treatment with the total push method. The larger group of 22 patients in a shorter period of time are making similar improvement in their social conduct and general reaction. They can be described as slightly improved, much improved and markedly improved. In none of these patients has the condition become worse. This improvement is shown in better contact with reality, in social responsiveness, increased activities, skill and coordination, sociability, improved mood and æstheticism with some degree of improvement in physical status. There has been a response to competitive games and to the reward-punishment and praise-blame concepts in accordance with the theories and principles advanced by Dr. Myerson. One of our patients, Patient 2, has improved to such an extent in his social status that a program of re-education has begun and he is already making satisfactory progress. It is our hope that such a program of re-education and an increasing program of psychotherapy may be applied to a considerable number of these patients.

We believe that if the insulin and metrazol therapy are used, the total push method should be instituted in its fullest sense as an important adjunct. We are offering it, however, as a distinct method of organized, intensive, individual activation in chronic schizophrenia. Such an intensive method of treatment has not only improved the mental and physical status of these patients, but since its success depends upon the thoroughly coordinated efforts of nurses, physicians, physiotherapists, physical instructors and occupational therapists, it has been stimulating to the entire personnel and has added much to the hospital morale.

FINGER AGNOSIA IN CHILDREN.

With a Brief Discussion on Defect and Retardation in Mentally Handicapped Children.*

By ALFRED STRAUSS, M. D.,† and HEINZ WERNER, Ph. D.‡

Finger agnosia is the inability to recognize, indicate on request, name or choose fingers of one's own hands or the hands of others. This disturbance, first described by Gerstmann in 1924, has been reported until the present time only in adult patients. It is the purpose of this paper to describe a similar syndrome found in mentally handicapped children. An attempt will be made to elucidate the difference of finger agnosia in children and in adults and to demonstrate the significance of the deficiency for mental growth.

History of the Problem.

In 1924, Gerstmann [6] of Vienna described a circumscribed disturbance of orientation in one's own body. The patient showed an isolated deficiency in the recognition of her fingers. She erred frequently when she was requested to touch, to point or to name the index, middle, ring or small finger or the thumb. Through a special effort, she sometimes succeeded in concentrating her attention upon her fingers. In 1927 and 1930, Gerstmann [7, 8, 9] added several new cases, some with presentation of autopsy findings. He regards finger agnosia, right-left disorientation, and pure agraphia as the essential elements of a syndrome with which acalculia is loosely associated. Since Gerstmann's contributions, many other cases of finger agnosia have been reported. (*Vide* bibliography.)

This syndrome may appear not only as a direct consequence of the circumscribed lesion; according to Gerstmann, finger agnosia comes about also during the period of restitution when a large

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area of the parieto-occipital lobe is involved. After restitution of gross defects, the differentiated symptom of finger agnosia may come to the fore. It is now generally believed that the parieto-occipital convexity of the left hemisphere represents what Klein calls "hand area"; the angular gyrus in its transition to the second occipital gyrus seems to be the region where the syndrome is specially localized.

Gerstmann interprets finger agnosia as a disturbance in the "body schema." By body schema is understood a spatial image which everyone possesses of his own body. It is a kind of diagram representing the single parts of the body in their spatial relation to each other. According to clinical investigations, kinaesthetic, tactile, and optical factors take part in building up the body image; impairment of any one of these factors may be predominant in the causation of disturbance in the body schema. Gerstmann holds the view that the fingers are a highly specialized part of the body schema, more vulnerable than other portions of the body. This hypothesis explains how finger agnosia can be present as a relatively isolated phenomenon without other disturbances in the body schema. The most differentiated parts of the hand area, the middle fingers, are most easily affected, and the thumb and little finger are least vulnerable.

Johannes Lange [16, 17] goes one step further in the interpretation of finger agnosia. He attempts to reduce this syndrome to a more fundamental impairment. According to him, finger agnosia is a disturbance not simply of the body schema as a subjective phenomenon, but it is an impairment of the ability to conceive of the hand as an articulated object in external space and to use it as an instrument for mental operations (arithmetic). On the basis of careful case-analyses, Lange contends that dedifferentiation of the hand area can be reduced to the more general impairment of a dedifferentiation of the structure of external space.

Paul Schilder [25] has made an attempt to demonstrate that the lack of recognition of the fingers can be found as a part of quite different clinical syndromes. He distinguishes five different types:

(a) Finger agnosia in its genuine sense is a specific disturbance of a part of the body schema, *i. e.*, the finger schema. It is the so-called Gerstmann's syndrome.

(b) Optic finger agnosia: the finger agnosia is definitely a part subordinated to a general syndrome of optic agnosia. (*Cf.* case of Schilder-Isakower.)[13]

(c) Constructive finger apraxia: although the patient is able to move intentionally or on command any of his fingers, he cannot copy the position of the fingers demonstrated to him. (*Cf.* Schlesinger's Case B.)[26]

(d) Apractic disturbance of the movement of the fingers. (*Cf.* case of Stengel-Schilder [24] and Schilder, Körperschema,[24] Cases 7, 8, 9, 10, and 11.)

(e) Finger aphasia: naming of the fingers is disturbed in consequence of a general impairment—amnestic aphasia.

After having finished our paper, the very interesting article by Nielsen [22a] on "Gerstmann Syndrome" appeared. The author reports several cases of finger agnosia and discusses the question of localization, particularly the relation of brain dominance, handedness and Gerstmann Syndrome.

Case Presentation.

J. B. Born 1-25-1922. Admitted to the Wayne County Training School June, 1935. Father died 1928 from tuberculosis. Mother remarried to a chiropractor; 42 years old; said to have had St. Vitus Dance at 15 years. She has been paralyzed on her left side since J.'s birth. She has lost considerable weight in the last two years. She is much interested in spiritualism, told the social worker that she expects to help J. through the messages she receives from his deceased father. Since January, 1938, the mother has been in a hospital because of a cerebrovascular accident, probably on basis of thrombosis of syphilitic nature.

Mother was in good health at the time the child was born. J. was born at full period; bottle fed; walked at about two years. Mother does not remember when he talked or cut his teeth. Whooping cough in 1928.

J. was examined first in the outpatient department of the Wayne County Training School at the age of 6 years 10 months. He was then in the first grade but not making any progress in school. He was reported as a disciplinary problem because he did not work and kept other children from work. The examination revealed a mental age of 5 years 6 months; I. Q. 80. During the psychological examination it was found that J. did not know the number of fingers on either or both hands. General behavior during the examination described as vivacious, good-natured, with good cooperation. Praised himself for succeeding with problems when he had actually failed. Reacted very promptly, giving little or no time to thought.

Physician's certificate for admission to the Wayne County Training School (April, 1935) states that J. was attending the sight saving class, 6-B, and

making very poor progress. I. Q. 65. Home situation very poor; boy not receiving proper training and supervision.

During the initial interview at the training school, J. showed fair insight for a borderline defective. He did not give the impression of an antisocial individual. Showed certain mannerisms which he interpreted as nervousness.

Physical Examination.—Undernourished, asthenic type. Septic tonsils. Some carious teeth. Eye examination (November, 1937): vision before refraction—right eye 20/200; left eye 20/200. Fundus examination showed an oval disc in each eye. After correction, vision—right eye 20/50 − 1; left eye 20/50. Diagnosis: mixed astigmatism, severe. Neurological examination (January, 1938): horizontal nystagmus to the left; sluggish reaction to light of the left pupil; both pupils react to light but not to convergence. Hyperactive tendon reflexes; Léri absent on the left arm; abdominal reflexes on the left side more hyperactive than on the right; Oppenheim reflex positive on the left foot. Slight hypoaesthesia on the right side with the exception of the corner of the mouth and the thumb (Goldstein's cortical sensibility disturbance?). Tonus: deviation in right arm more than in left, both inwards; deviation (Hoff-Schilder) in left arm upwards, in right arm downwards. Joint sense and stereognostic sense normal. Tactile localization on both hands impaired; on the right side more than on the left. Laboratory findings: Kahn negative on both blood and spinal fluid. In spinal fluid: Pandy reaction negative; Nonne reaction negative; cells none. Clinical diagnosis: a diffuse cerebral process.

Binet Examinations.—August, 1935: Chronological age, 13 years 7 months; mental age, 9 years 4 months; I. Q. 69. August 1937: chronological age, 15 years 7 months; mental age, 9 years 10 months; I. Q. 63.

Stanford Educational Achievement Test.—Reading (average), 10 years 11 months, grade 5; arithmetic (average), 8 years 8 months, grade 3.1. Educational quotient 65.

Stenquist Assembling Test.—Approximately at the median for his chronological age, 14 years 6 months.

Cottage Reports.—Well adjusted.

Clinical Analysis.—J. B. was brought to our attention in the course of a study on arithmetic disability. The teacher referred him to us as one of the extreme cases of arithmetic deficiency. We found that he has considerable difficulty in counting with his fingers. When he was asked with closed eyes to indicate a number by using the fingers on one hand, he succeeded. Demonstration of numbers beyond five by using both hands was difficult; he counted one by one and still made errors. His difficulty in counting by fingers led us to test him on finger agnosia. The examinee was tested through out with his eyes closed; this is at variance with the usual procedure observed in testing adult patients and will be discussed later. He was examined in the following test situations:

1. Showing one or two fingers of one hand as they were named.*
2. Pointing on command at one or two fingers of one hand.

* He was to name the fingers by numbers, beginning with the thumb as one and ending with the little finger as five.

3. Naming one or two fingers of one hand which had been touched by the examiner.

4. Pointing out one or two fingers of one hand which had been touched by examiner.

5. He was asked to place his right (left) hand on the table opposite the right (left) hand of the examiner; he had to indicate on the examiner's hand which of his own fingers, or which two, had been touched.

6. A picture of a right (left) hand was drawn on the blackboard. With eyes closed the boy was touched; after having opened his eyes, he had to indicate on the picture which of his fingers (or two fingers) had been touched.

7. Pointing out the two fingers, each on different hands, which had been touched successively by the examiner.

8. He was asked to close his eyes and make a fist. The examiner took hold of the fist and straightened a certain number of fingers. The boy had to tell how many fingers had been straightened.

He made errors in all tests. The errors increased from tests 1 through 6. He made more errors in all tests in which two fingers rather than one were involved. He was better with the thumb and little finger than with the middle fingers. In tests 7 and 8, he failed completely.

To answer the question whether or not such a deficiency as that shown by J. B. is dependent on mental age, we examined with the above tests a group of 30 children, ranging in mental age from seven years to eleven years eight months. It was found that no correlation exists within the range examined between this deficiency and the mental age.

Bearing in mind the clinical analyses by Lange and others [5, 14–17, 22, 25, 28] who tried to link this disturbance of the finger schema with an impairment of a more general nature, we presented J. B. with problems involving the grasp of spatial relationship. We chose simple situations with which his astigmatism did not interfere. It has been found that this boy has a severe impairment in grasping spatial relations and in constructive ability.*

The boy shows a marked arithmetic disability. We know from cases reported in the literature that finger agnosia is frequently associated with arithmetic deficiency, so-called acalculia. Space does not permit discussion of the entire problem of acalculia. We are concerned here with acalculia only as a symptom of retardation in mental development and insofar as it is related to finger agnosia. It seemed, therefore, expedient to recur to selected groups of children: one group known to be relatively outstanding in arithmetic achievement, the other group demonstrating considerable retardation in the development of number concept. Fourteen children of each group were tested with a battery of 13 test items concerning the finger schema. In the arithmetic ability group only three children made (a few)

* *Cf.* our detailed report in "Problems and Methods of Functional Analysis in Mentally Deficient Children (Illustrated by Cases of Arithmetic Disability)." Journal of Abnormal and Social Psychology.[38]

errors. In the arithmetic disability group there was only one boy who made no errors at all, and eleven of the boys made a considerable number of errors.*

Summary.

A 16-year-old boy shows somatically a diffuse cerebral process. With a mental retardation of 5 years 9 months, a retardation in reading of 4 years 5 months and in arithmetic of 6 years 8 months, this boy has a definite defect in the finger schema. In addition, he shows a disability in the grasp of spatial relations. A comparative investigation, using other mentally deficient children with relatively either exceptionally high or low arithmetic achievement, gave evidence of a relation between deficiency in the finger schema and a retardation in the development of number concept.

Theoretical Discussion.

1. The first point of our discussion concerns the *definition* of finger agnosia. Is one entitled to use the term finger agnosia in cases of this kind? Finger agnosia has been defined in the literature as a lack of recognition of the fingers with the eyes open. Our cases showed this deficiency only with the eyes closed. According to the current definition, agnosia is an inability to work up conceptually data coming from normally functioning sense organs.[12] It seems, therefore, justified to call the disturbance found in our children "finger agnosia."

2. A further question refers to the essential differences between finger agnosia in the *child* and in the *adult*. The fundamental difference between a brain defect in the child and a cerebral lesion in the adult is: in the child, the defect hinders more or less the development of certain abilities; in an adult, the lesion destroys or disturbs certain factors in mental operations already fully developed. (Destruction of the tissue in the speech region produces mutism in the young child; a similar lesion occurring in an adult occasions only a dysfunction of speech.) This general principle applies to finger agnosia in particular. It seems, therefore, obvious that in adults finger agnosia is only loosely associated with acal-

* *Cf.* our paper: Deficiency in the Finger Schema in Relation to Arithmetic Disability. Amer. Journal for Orthopsychiatry.[31]

culia, while in our cases a definite relation exists between both symptoms. Inquiring into the possible causes of such a difference, one might start with the hypothesis of Lange and others that finger agnosia as a disturbance in the body schema means a loss of an articulated instrument for mental operations, *e. g.*, as used in building up number concepts. We may assume that *the primary number concept exists concretely as a configuration of the fingers of the hand.* If such an articulated schema cannot be formed, a basic element for a natural development of the number concept is absent. This assumption may also serve to explain the much looser relationship between finger agnosia and arithmetic deficiency in the adult. For the adult, number operations have usually reached a stage where the concrete configurations are replaced by abstract number forms and where, therefore, the articulated hand as a tool for these mental operations is hardly needed. One may expect that acalculia will appear in finger agnostic patients, particularly if they are either individuals of a rather primitive mental structure or persons of a higher level who had been accustomed to link their mental operations, to a considerable degree, with concrete schemata.

3. Since finger agnosia has been demonstrated in mentally deficient children, first in a rather extreme single case and then in a group of individuals showing this impairment in a lesser degree, the question seems unavoidable as to the relation between *defect* and *retardation.* Roughly speaking, a defect can be genetically defined as an arrest of the growth of a function, a retardation as a delay in its normal development. The actual knowledge which we have today on this point is very scant indeed. There are, nevertheless, good reasons which seem to warrant a brief discussion. If a definite distinction between defect and retardation could be demonstrated on the basis of clinical-psychological analysis, one would have an important tool for a refined classification as to the difference between primary and secondary amentia (Tredgold [32]) or endogenous and exogenous mental deficiency (Strauss [29]); this distinction would also enable us to make definite prognoses and differentiated programs for reeducation. A distinction between defect and retardation should be psychologically defined by three characteristics:

(a) Different individuals characterized as retarded in some particular field may demonstrate quite different symptoms of the dis-

turbance; in the case of a defect all these symptoms and perhaps some additional symptoms should appear collectively in one individual.

(b) There should be, at least partially, a qualitative difference in the type of errors made.

(c) An indirect demonstration of the difference would be that educational methods successfully used in retarded children are not applicable for overcoming the defect.

Illustrations to point a and b may be furnished from our case analysis. J. B. showed errors with every single item of the finger schema test; the individuals of the arithmetic disability group failed only in a few. J. B.'s score of errors is far in excess of the average score of the group. Moreover, the errors made by J. B. point to a higher degree of the disturbance than was present in the individuals of the group. He failed in test situations where only one finger was involved, while nearly all individuals of the group were able to pass this test without errors.

This failure with only one finger leads us to the qualitative aspect of the difference between his performance and the performance of the other boys. When presented with the task of pointing fingers, each on different hands, touched previously by the examiner, J. B. made more errors than in an analogous task where both fingers touched belonged to the same hand. This result is in striking opposition to the behavior of the boys of the arithmetic disability group; these boys scored better when the two fingers touched successively belonged to different hands. The difference in the results can be interpreted only as qualitative in nature.

To explain these differences still further, one may advantageously recur to a concept of gestalt-psychology, *e. g.*, the concept of "foreground (figure) and background." In the case of J. B. where one finger cannot be conceived as an articulated "figure" against the hand as a "background," the result is a diffuseness of the hand area as a whole. If touched, therefore, on two fingers of the same hand successively, one might expect him to improve because of greater accentuation of the foreground. An improvement may not, of course, come into appearance because of the greater complexity of the task itself, *i. e.*, discerning two fingers in succession instead of one. When touched on two fingers, each on different hands, he failed completely because he faced now two

diffuse fields, doubling, as it were, the difficulty which he already had in the one finger test. The boys of the group, however, not having any difficulty in the one finger experiment showed less errors with two fingers of different hands compared with the test where both fingers of the same hand had to be pointed out. We may assume that these boys conceive of their fingers as being a figure (however blurred their image of the fingers may be) detached from the background of the hand. For those children whose difficulties lie only in the indistinctness of the relation of the fingers to each other, the task should be greatly facilitated by presenting to them an objectively more articulated field (two hands, one finger each, as against two fingers on the same hand).

To point c: finally we would like to mention that, on the basis of our results, a program of remedial treatment is in preparation. The general principles on which we have started the program may be briefly discussed. One of the fundamental questions is that of remedial teaching as related to the difference between defect and retardation.[4, 30] There is a widespread trend in classes for mental defectives towards utilization of the methods with which normal children are taught. These methods cannot be applied to the particular mental structure of children with a special arithmetic disability. The so-called sense training (Montessori) is too elementary in its scope to be used in cases with an impaired function of a complex nature. We believe that the general concept of the methods of Decroly and Descoeudres [4] has been most fruitful for developing a training program for our own cases. Having the advantage of dealing with cases already analyzed as to their particular dysfunction, we are enabled to go one step beyond the educational goal set by Decroly and to formulate educational devices emerging from our knowledge of the functional pattern. Our remedial work is aiming at the particular deficiency, *e. g.*, the diffuseness in the finger configuration; it attempts to build up distinct relations of the elements (fingers) to each other and to the whole in view of the significance of these relations to the concept and system of numbers.

We do not expect that a method planned beforehand and useful for the individuals with arithmetic retardation would succeed in all cases, particularly not in those with a defect. Since it is our

experience that the pedagogical situation in retarded and in defective children is fundamentally different,* clinical analysis and educational guidance have to be strongly interlinked in cases of defect so that the pedagogical situation becomes an intimate part of psychological experimentation.

BIBLIOGRAPHY.

1. Angyall, L. V.: Beitraege zur Symptomatologie, Lokalisation und hirnpathol. Auffassung des Gerstmann'schen Syndroms. Z. f. d. ges. Neur. u. Psych., 156: 245, 1936.
2. Buerger-Prinz, H.: Ueber eine Stoerung des Rechnens. Nervenarzt, 8: 586, 1935.
3. Conrad, K.: Versuch einer psychologischen Analyse des Parietalsyndroms. Monatsschr. f. Psych. u. Neur., 84: 28, 1933.
4. Descoeudres, A.: The education of mentally defective children (George G. Harrap & Co., London, 1928).
5. Ehrenwald, H.: Stoerungen der Zeitauffassung, der raeumlichen Orientierung, des Zeichnens und des Rechnens bei einem Hirnverletzten. Z. f. d. ges. Neur. u. Psych., 132: 518, 1931.
6. Gerstmann, J.: Fingeragnosie. Eine umschriebene Stoerung der Orientierung am eigenen Koerper. Wien. klin. Woch., p. 1010, 1924.
7. ———: Fingeragnosie und isolierte Agraphie—ein neues Syndrom. Z. f. d. ges. Neur. u. Psych., 108: 152, 1927.
8. ———: Zur Symptomatologie der Hirnlaesionen im Uebergangsgebiet der unteren Parietal-und mittleren Occipital-windung. Nervenarzt, 3: 691, 1930.
9. ———: Zur Symptomatologie der Herderkrankungen. D. Z. f. Nervenh., 116: 214, 1930.
10. ———: Ueber Fingeragnosie. Jahrb. f. Psych., 48: 135, 1932.
11. Guttmann, E.: Congenital arithmetic disability and acalculia (Henschen). Br. J. Med. Psychol., 16: 16, 1936.
12. Handwoerterbuch der medizinischen Psychologie (ed. Birnbaum). Georg Thieme, Leipzig, 1930.
13. Isakower, O., and Schilder, P.: Optisch-raeumliche Agnosie und Agraphie. Z. f. d. ges. Neur. u. Psych., 113: 102, 1928.
14. Klein, R.: Ueber die Funktionen des Parietallappens. Nervenarzt, 6: 1, 1933.
15. ———: Ueber ein parietales Stoerungssyndrom. Z. f. d. ges. Neur. u. Psych., 160: 417, 1937.
16. Lange, J.: Fingeragnosie und Agraphie. Monatsschr. f. Psych. u. Neur., 76: 129, 1930.

* To illustrate, the reader may recall that the usual methods for reading disability do not bring progress in cases of alexia.

17. ———: Probleme der Fingeragnosie. Z. f. d. ges. Neur. u. Psych., 147: 594, 1933.
18. Laubenthal, F.: Zur psychologischen Analyse von Kranken mit einem Syndrom der linken Parieto-occipitalgegend. Arch. f. Psych., 99: 147, 1933.
19. Lindqvist, T.: De l'alcalculie. Acta med. scand., 87: 224, 1935.
20. ———: Nouvelles études sur le problème de l'alcalculie. Acta med. scand., 88: 217, 1936.
21. Marburg, O.: Scheitellappenerweichung unter dem Bilde eines Pseudotumors cerebri (zugleich ein Beitrag zur Frage der Fingeragnosie ohne Agraphie). Arb. neurol. Inst. Wien, 33: 1, 1931.
22. Muncie W.: Fingeragnosia (Gerstmann). Bull. Johns Hopkins Hosp., 57: 330, 1935.
22a. Nielsen, J. M.: Gerstmann Syndrome: Finger Agnosia, Agraphia, Confusion of Right and Left and Acalculia. Arch. Neur. Psych., 39: 536, 1938.
23. Poetzl, O., und Hermann, G.: Ueber die Agraphie und ihre lokaldiagnostischen Beziehungen. Monograph. S. Karger, Berlin, 1926.
24. Schilder, P.: Das Koerperschema. J. Springer, Berlin, 1923.
25. ———: Fingeragnosie, Fingerapraxie, Fingeraphasie. Nervenarzt, 4: 625, 1931.
26. Schlesinger, H.: Zur Auffassung der optischen und konstruktiven Apraxie. Z. f. d. ges. Neur. u. Pysch., 117: 649, 1929.
27. Singer, H. D., and Low, A. A.: Acalculia (Henschen). Arch. Neur. & Psych., 29: 467, 1933.
28. Stockert, F. G.: Das Gerstmann'sche Syndrom der Fingeragnosie. Monatsschr. f. Psych. u. Neur., 88: 121, 1934.
29. Strauss. A.: Beitraege zur Einteilung, Entstehung und Klinik der schwersten Schwachsinnsformen. Arch. f. Psych., 9: 693, 1933.
30. ———: Pedagogía terapéutica. Editorial Labor, Barcelona, 1936.
31. ———, and Werner, H.: Deficiency in the Finger Schema in Relation to Arithmetic Disability (Fingeragnosia and Acalculia) Amer. J. Orthopsych., 8: 719, 1938.
32. Tredgold, A. F.: A textbook of mental deficiency. 6th edition. Baltimore, 1937.
33. Werner, H., and Strauss, A.: Problems and methods of functional analysis in mentally deficient children. J. Abn. Soc. Psychol. 34: 37. 1939.

PSYCHIATRIC FACILITIES IN CHICAGO.

COMPILED BY THE ILLINOIS SOCIETY FOR MENTAL HYGIENE.

The following information is taken from the Handbook of Information, which has been compiled periodically by the Illinois Society for Mental Hygiene since 1932. The Handbook contains data of the hospitals and institutions in the state of Illinois, which are operated "not for profit," and which are licensed by the State and registered by the American Medical Association.

The Illinois Society for Mental Hygiene, under the direction of Dr. Conrad S. Sommer, located at 203 North Wabash Avenue, is a private agency supported in part by the Chicago Community Fund and in part by memberships and contributions. Its facilities, which include a library, are available to social agencies and individuals in a consultation and advisory capacity. Training courses in social service are conducted, and the Society makes available information concerning clinics, hospitals, rest homes and psychiatric services generally. Discussion groups are maintained and lecture programs provide for popular education in the field of mental hygiene.

Central Free Dispensary, Psychiatric Clinic, 1748 West Harrison Street, is under the directorship of Dr. Peter Bassoe. There is a staff of psychiatrists, psychologists and a social worker, who conduct a diagnostic clinic on Tuesdays and Wednesdays. A children's clinic is also held weekly at the dispensary.

Central Free Dispensary, Behavior Clinic, 629 South Wood Street. Psychiatrist—Dr. Ralph C. Hammill. This clinic deals with behavior problems of all types in children and young persons up to the age of twenty.

The Douglas Smith Fund, 844 Rush Street. The Douglas Smith Health Service offers complete health examination to young women sixteen to thirty-five years of age who are employed or employable but unable to pay a private physician for comparable service. Psychiatric examination is also provided on recommendation of the examining physician.

Loyola University School of Medicine, Division of Neuropsychiatry, Adult Psychiatric Clinic, 706 South Wolcott Avenue. The medical director is Dr. Francis J. Gerty and there is a staff of psychiatrists, psychologists and psychiatric social workers. Patients may be referred to the clinic by any responsible agency and are received for ambulant care only.

Loyola Mercy Child Guidance Clinic, 2526 Calumet Avenue. This clinic deals with cases referred by parochial or public schools, social agencies and individuals.

Mandel Clinic of Michael Reese Hospital, 29th Street and Ellis Avenue. Director—Dr. Jacob Kasanin. This clinic maintains a staff of seven psy-

chiatrists, two fellows in psychiatry, four psychiatric social workers and five psychologists. Clinic services for both children and adults are by appointment only.

Max Epstein Clinic of Provident Hospital, 5034 Vincennes Avenue. This clinic staffed by psychiatrists, social workers and a psychologist deals with patients on the appointment basis.

Mount Sinai Hospital Dispensary, 2750 West 15th Place. There is a staff of eight psychiatrists and a social worker in this clinic, which is conducted under the direction of Dr. Meyer Solomon. The clinic is conducted twice weekly.

Northwestern University Medical Clinics, 747 Fairbanks Court. Chief of staff is Dr. Lewis J. Pollock, and there are twenty-four psychiatrists, a psychologist and a social worker attached. Clinics are held daily for both adults and children.

St. Luke's Hospital Neuropsychiatric Clinic, 1440 Indiana Avenue. Six psychiatrists are available and patients are seen by appointment.

University of Chicago, Division of Psychiatry, University Clinics, 950 East 59th Street. Clinic Director—Dr. David Slight. Services for adults and adolescents are provided at Billings Hospital, and for children at Bobs-Roberts Hospital.

Women and Children's Hospital Clinic, 138 North Ashton Avenue. Neurological and psychiatric clinics are held twice weekly.

Criminal Court, Behavior Clinic, 2600 South California Avenue. This clinic with a staff of three psychiatrists, a social worker and psychologist, provides advisory psychiatric service to the criminal courts. Cases are received only by order of the judge in the case, or of the chief justice of the criminal court.

Juvenile Court Branch of the Institute for Juvenile Research, 2246 West Roosevelt Road. Cases are received for examination when referred by the judge of the Juvenile Court.

Municipal Court, Psychiatric Institute, 1121 South State Street. Director—Dr. D. Rotman. The Institute serves as psychiatric consultant to the judges of the Municipal Court.

Bureau of Child Study, Board of Education, 228 North LaSalle Street. Director—Dr. Grace Munson. The Bureau staff includes a medical examiner, psychologists, educational experts, a psychiatrist and a speech pathologist. Psychological services are provided for school children of all levels.

Chicago State Hospital, Clinics for Patients on Parole, 6400 Irving Park Road. Regular clinics are conducted by pyschiatrists from the Chicago State Hospital, the Elgin State Hospital, the Kankakee State Hospital and the Manteno State Hospital for parole patients from those institutions.

The Chicago Association for the Study and Advancement of Individual Psychiatry, Child Guidance Clinic, 612 North Michigan Avenue. Sessions are held Friday afternoons, primarily for parents and teachers.

The Institute for Juvenile Research, 907 South Wolcott Avenue. Director—Dr. Paul L. Schroeder. The Institute is a part of the Division of the

Criminologist under the State Department of Public Welfare. Its purpose is the study of behavior difficulties of children and their treatment indications. The service is state-wide and community clinics are held periodically in the larger cities of the state. The age limits are eighteen years for girls, seventeen years for boys.

The Institute for Psychoanalysis, 43 East Ohio Street. Director—Dr. Franz Alexander. The Institute is interested primarily in research and training. There are nine physicians on the staff. Hours are by appointment only.

Northwestern University College Student Counseling, 301 East Chicago Avenue. Dr. Minna Emch conducts this service, which is for students in University College only.

University of Chicago Student Health Service, 950 East 59th Street. Dr. Douglas G. Campbell and Dr. Charles B. Congdon conduct this service for students at the University of Chicago only.

Chicago State Hospital, 6400 Irving Park Road. Managing officer—Dr. Edward F. Dombrowski. This is one of the large state institutions with a bed capacity for 4288 patients.

Psychopathic Hospital, Polk and Wood Streets. Medical director—Dr. Francis J. Gerty. Bed capacity 175. This hospital, a division of Cook County Hospital, is primarily a diagnostic centre for patients who are to have a hearing before the Sanity Commission of the Cook County Court. The average hospital residence is six days.

University of Illinois, Psychiatric Institute, 1819 West Polk Street. Director—Dr. H. Douglas Singer. Number of beds 60. The Institute serves the three-fold purpose of teaching, therapy and research. As a rule it receives only patients committed by the court. An out-patient service is conducted daily except Saturday and new patients are seen by appointment only.

WILLIAM ALANSON WHITE.

UNIV. OF MICH.

Comment.

DEDICATION OF PORTRAIT OF DR. WILLIAM ALANSON WHITE.

On January 31, 1939, at Saint Elizabeths Hospital, Washington, D. C., assembled a large and distinguished audience on the occasion of the presentation of a portrait of the former superintendent, Dr. William A. White.

This gift to the hospital, made possible by the cooperation of the friends and former associates of Dr. White, including many of the physicians and employees of the institution he presided over with distinction for so many years, is the work of the portrait painter Mr. Henry R. Rittenberg. Readers of the JOURNAL will welcome its reproduction herewith.

The formal presentation was made by Rev. Ulysses G. B. Pierce, president of the Board of Visitors, and was accepted on behalf of the hospital by the Hon. Harold L. Ickes, Secretary of the Interior.

Saint Elizabeths Hospital was opened in 1855, and Dr. White was its fourth superintendent. In his address Secretary Ickes referred to the history of Saint Elizabeths and the part Dr. White played in its development.

The hospital was established as a bureau of the Department of the Interior and it has always been a credit to the Department in its enlightened dealings with the more than forty-four thousand patients, drawn chiefly from the Army, the Navy and the District of Columbia, who have passed through its doors since it was established as the result of the efforts of that great woman, Dorothea L. Dix.

Dr. White's administration was the longest, and in many ways the most significant. He came to Saint Elizabeths in 1903 when the hospital had only about 2300 patients. A large building program was then under way, and it was indeed fortunate that he was young, well trained and full of enthusiasm when he took over his task. Mental institutions at that time were, to a large extent, cus-

todial. They were also, unfortunately, often the objects of suspicion in the public mind. Psychiatry was still in the groping stage and had hardly begun to emerge into its present dignity as a specialty of medicine.

Dr. White immediately undertook to improve the care of the patients by furnishing increased individual attention and by perfecting the training of the medical staff and the ward personnel. It was his aim to make this a curative institution rather than a domiciliary one. As a matter of fact, for every one hundred patients admitted, of recent years 45 or more have been discharged, most of them as recovered or as much improved; a much higher proportion than is generally assumed by the public.

He found in existence in the District of Columbia a barbarous and antiquated method of commitment of patients, by which a jury trial in a criminal courtroom was compulsory. He protested against this system, in season and out, building up a public opinion which finally forced a change in the law, but only after his death. It is tragic that Dr. White could not have lived to see the accomplishment of this great humanitarian effort.

Dr. White was an eloquent speaker and writer who devoted much of his attention to the task of interpreting psychiatry to both the medical profession and the public. He was professor of psychiatry at George Washington University from the time that he came to Washington, and for many years he taught also at Georgetown University as well as at the Army and Naval Medical School. By his books, his articles in medical journals and his lectures, he did more than any other man in this country to bring psychiatry to its present place of recognition as an integral and important part of medicine. To the general public he preached that mental disorder is a natural phenomenon, and that the mental hospital is properly entitled to the same emotional recognition and appreciation as the general hospital.

In the operation of the institution he constantly sought to take advantage of new methods of treatment and of administration for the benefit of the patients. Saint Elizabeths was the first hospital in this country to adopt the malarial treatment for general paresis, now recognized as one of the most significant advances made in therapy in the last generation. Dr. White set up the first separate medical and surgical service in a mental institution, a

practice which is now finding many supporters. He was an early exponent of the theories of Freud and advocated them fearlessly at a time when there was intense opposition to them on the part of many psychiatrists. The wisdom of his course is demonstrated today by the widespread acceptance of these teachings which were once anathema to so many of his colleagues.

It was by Dr. White's effort that the name of the institution was changed in 1916 from "Government Hospital for the Insane" to Saint Elizabeths Hospital. He had long felt that the word "insane" in the title of the hospital had a bad psychological effect upon the patients and their relatives. This change was indeed a happy one and did much to overcome the supposed stigma resulting from care in the institution.

During Dr. White's long administration of thirty-four years the hospital grew constantly until it came to be widely recognized as perhaps the outstanding mental hospital of the world. Today it is cited as a model for others to follow. Physicians trained at Saint Elizabeths are in demand from other mental hospitals throughout the country. At the time of his death Dr. White was considered the outstanding psychiatrist of America, and Saint Elizabeths Hospital is fortunate to be the recipient of much of the glory which has been reflected from its illustrious superintendent.

Dr. Winfred Overholser, the present superintendent of Saint Elizabeths Hospital, who presided at the meeting, made the announcement that the valuable medical and psychiatric library which Dr. White had accumulated through the years had been generously offered by Mrs. White as a gift to the hospital to be suitably housed and maintained as the William A. White Memorial Collection.

CHARACTERISTICS OF THE DRUG ADDICT.

The results of a statistical analysis of the clinical records of 1036 drug addicts admitted as patients to the United States Service Hospital at Lexington, Ky., during the fiscal year July 1, 1936, to June 30, 1937, Dr. Michael J. Pescor, Passed Assistant Surgeon, U. S. Public Health Service, summarizes in an unusual and interesting way. He describes a "statistical addict" who is a composite of averages and highest frequencies in the total material. This

description, which concludes a report in Supplement No. 143 to the Public Health Reports, is as follows:

Such a hybrid individual would be a white male prisoner, 38 years of age, given a 2-year sentence for the illegal sale of narcotics by a federal court. His family history would be positive for such familial diseases as cardiac disease, tuberculosis, or cancer, and if any psychopathic determinants existed they would most likely be alcoholism or drug addiction. His parents would be in marginal economic circumstances, average disciplinarians, and the family relationships would be congenial. The patient would be one of several children, a native of native parentage, the parental home would be intact up to the age of 18 years, and the childhood adjustment would apparently be normal. He would be brought up in religious faith, but would discontinue church affiliations as an adult. He would graduate from the eighth grade, taking up an occupation classified in the domestic and personal service. As an adult he would live in a deteriorated metropolitan section. More than likely he would have to resort to illegal means of earning the additional income required to support his drug habit. He would marry, but his marriage would probably terminate in separation or divorce. He would have no children, possibly because drugs deprived him of a normal sexual urge. He would probably make a satisfactory social adjustment prior to addiction, but not after addiction. He would be tolerant toward all forms of vice, occasionally indulging in all forms. He would not give a history of military service.

He would become addicted to morphine at the age of 27 through the influence of associates and curiosity. He would use more than one narcotic drug, but would prefer morphine when it was obtainable. The last drug used would, therefore, most likely be morphine. He would be addicted about 10 years. He would probably give no history of voluntary attempts at cure, would admit at least one enforced treatment in a jail or penitentiary, but would not remain abstinent any longer than 2 years at the most, relapsing because of association and desire to recapture the pleasant sensations produced by drugs.

His first arrest would occur at the age of 28 for violation of drug laws for which he would have an equal chance of being acquitted or sent to the penitentiary. He would not have a delinquency record prior to addiction. After addiction his offenses would more than likely be confined to violation of drug laws for which he would be given at least one penitentiary sentence and at least one jail sentence. He would probably have spent a total of 3 years behind bars on previous sentences.

He would give a history of the usual childhood diseases without complications, but as an adult he would be subject to some chronic disease such as heart trouble, arthritis, tuberculosis, or asthma. He would deny any mental disorders, but if he did admit any it would be a tendency toward neurosis. However, he would readily admit a history of gonorrhea. Ninety-nine chances to one he would have poor dentition, either caries or pyorrhea alveolaris; there would be a strong possibility of defective vision also. However, his physical defects would not prevent him from doing manual labor. The psy-

chologist would probably give him the Army Alpha test, which would disclose that the hypothetical patient had a mental age of 13 years, 8 months. The psychiatrist would give him a classification of psychopathic diathesis, which means that the patient has an uncrystallized personality defect and that he became addicted through the desire to seek new thrills.

During his stay in the institution he would abide by the regulations, show a good knowledge of his occupational assignment, and would be a willing worker. He would be accepted by his fellow patients and would like to work with them. The custodial officers would find him pleasant and agreeable. As the time for his release approached he would maintain that he was through with drugs forever because he did not want to spend the rest of his life in jail, indicating that he still thought drugs were beneficial, but the penalty outweighed the benefit. He would plan to live with responsible relatives largely at the insistence of the hospital officials. However, he would have no offer of employment to look forward to. He would be given an average prognosis for permanent cure which is a vague way of stating that he will probably relapse.

MEDICAL EDUCATION.

Preliminary steps have been taken in the formation of an advisory council on medical education, licensure and hospitals, and the following organizations were invited to attend a conference to discuss the formation of such a council on February 11, 1939:

	No. of Representatives.
Association of American Medical Colleges	3
American Medical Association	3
American Hospital Association	3
Federation of State Medical Boards	3
Advisory Board of Medical Specialties	3
National Board of Medical Examiners	1
American College of Surgeons	2
American College of Physicians	2
Association of American Universities	2
American Public Health Association	1
American Association for the Advancement of Science (Division of Medical Sciences)	1
	24

The American Board of Psychiatry and Neurology was represented by Dr. Franklin G. Ebaugh, who served as chairman of the group appointed by the American Board of Medical Specialties which is composed of representatives of various specialty boards.

A large number of organizations are dealing with various features of medical education, licensure and hospitals in this country, some of them with conspicuous success in their own spheres of influence. At the same time there is a great deal of duplication, overlapping, competition and confusion. Also there are important needs that no individual agency is covering satisfactorily and for which no organization feels responsible. It is of great importance to the public and to the profession that the educational phases of medicine be integrated and that existing programs be adapted to meet more effectively the responsibilities which are likely to be placed upon medicine in the future.

Medical training now has become primarily the responsibility of medical schools and hospitals. All students today must have a period of college preparation preceding the professional training; the number of medical schools has been greatly reduced, yet the number of graduates is almost as high as ever and there are a number of weak institutions still in existence; nearly every graduate takes a hospital internship; every state has its own agency for licensure; the new nation wide plans for graduate and postgraduate education involving the cooperation of the medical schools, hospitals and the profession have been instituted. These developments have occurred in segments and sometimes without much relationship to other parts of the whole structure of medical education or to the programs of other agencies carrying out parallel or duplicating activities.

Full credit is due the various individuals and organizations which contributed to the rapid elevation of the standards of medical training in this country, particular to the courage, leadership and financial aid of the universities and educational foundations, the participation of hospitals in the teaching plans, and the enforcement of standards by the state boards. Special credit should be given to the Council on Medical Education and Hospitals of the American Medical Association in dealing with the proprietary, commercial and weaker schools and in the collection and distribution of data on students, schools, state board activities, and other features of the whole program. It is now realized widely that the evaluation of the objectives and programs of medical training broadly conceived can best be secured not by an agency representing the profession alone, which really is the alumni body of the schools, but

by one which represents fully as much the educational, hospital, licensing and other phases of the whole problem.

The internship, as one example of the problems involved, has become universally recognized as an essential part of the basic preparation for practice. Twenty states now require such a training for admission to the licensing examinations. Some of them so define and regulate this period of training, however, that they defeat the efforts of universities, medical schools and hospitals to provide a satisfactory preparation adapted to the needs of those going into different fields of practice. Uniformity, rigidity and regulation are not distinquishing characteristics of an educational program. It is well known that the intern period is poorly adjusted in many instances to the preceding medical course, to the needs of the student and to subsequent graduate training. Even the approval of national accrediting bodies is uncertain. An intensive study of internships in a group of eastern hospitals recently has shown that not more than one-half of the hospitals approved for intern training by the Council on Medical Education and Hospitals provides satisfactory educational standards. The proportion of residencies meeting a real educational level is smaller. There is need for joint and continuing study of the place and functions of the internship and residency in the evolution of the medical course, graduate training and licensure.

Plans already developed for graduate and postgraduate training will require wide and, in some instances, fundamental readjustments in the basic medical course and in hospital services. The medical schools and universities are being called on to assume responsibilities in these programs. All are being subjected to numerous surveys and inspections by different agencies, frequently overlapping in their interests and conflicting in their objectives. The 12 American specialty boards and the Advisory Board of Medical Specialties, created in 1933, recognize the dangers of rigidity, regimentation and regulation in a field of professional training which must depend so largely upon the educational initiative, self-reliance and resourcefulness of the individual. The problems from college preparation to retirement from professional life should be looked upon now as parts of a single educational program. Portions of the program are primarily within the jurisdiction of universities, some are largely within the domain of the hospitals,

others are in the various fields of practice, and some are under governmental regulation.

It is becoming increasingly apparent that there is need of coordination of the various phases of medical education and better definition of the several areas of responsibility of national and state agencies, universities, hospitals and professional bodies dealing with portions of the whole program, if medicine in this country is to meet fully its obligations. The logical conclusion from the present more or less unrelated and frequently overlapping efforts is to create a national coordinating body representative of the major activities in medical education and service in order more effectively to meet the new conditions and needs of the country. No agency exists now that can fill this need.

A National Council on Medical Education, Licensure and Hospitals should be created from within the present organizations, made up of representatives of the universities, medical schools, hospitals, practicing profession, specialty boards, state licensing bodies and public health agencies. The functions of such a National Council would include the study and evaluation of the major educational needs of American medicine, mobilizing the best current opinions regarding the different phases of professional training at its several levels, formulating adequate standards for these activities, and advising regulatory bodies and governmental agencies on standards, methods, procedures and areas of action. The National Council should delegate to existing organizations all administrative functions and endeavor to coordinate the efforts and simplify the procedures of the multiple agencies now in operation. A central clearing house, carrying influence and prestige by virtue of the knowledge and judgment of its personnel and the organizations which it represents and providing a suitable vehicle of our own creation for cooperation on matters dealing with all features of medical education, transcending the activities and interests of any single group or organization, would be of the greatest practical value to the profession, the universities, the hospitals, the licensing bodies and the future health program of the entire country. The cooperation of the medical schools, hospitals, licensing boards and those professional organizations which have considered the undertaking has already been assured.

News and Notes.

MICHIGAN NEUROPSYCHIATRIC INSTITUTE.—The Neuropsychiatric Institute of the University of Michigan Hospital was dedicated on February 11, 1939. Dr. Alexander G. Ruthven, president of the University of Michigan, presided over the exercises which were attended by members of the faculty, physicians, legal authorities and social workers. The principal speaker was Dr. Rock Sleyster, president-elect of the American Medical Association, who discussed modern trends in mental hygiene and the aims of such an Institute.

The newly completed Institute is an integral part of the University Hospital and has a bed capacity of 82. Of these, 61 beds are available for adults and 21 for children. The building has five floors, four of which are devoted to the care of in-patients and include a gymnasium, hydrotherapeutic suites and occupational and recreational therapy units. An out-patient clinic is conducted by the staff of the Institute. This is available for consultation service to other clinics, for the treatment of out-patients and examinations preliminary to in-patient care.

The physical continuity of the main hospital and the Neuropsychiatric Institute will facilitate a ready exchange of consultation services and assist in a free interchange of ideas and clinical approaches. In addition, it will provide a wealth of unusual clinical material for research and teaching. All types of laboratory services are available including a complete neuropathological division in the Institute.

The Institute will provide facilities for the treatment of incipient or early mental disorders. It will provide a diagnostic service as well as assistance and recommendations to legal authorities and social agencies. Patients are accepted for hospital treatment only by voluntary admission although certain selected cases may be transferred from state hospitals.

AMERICAN PHYSICIANS' ART ASSOCIATION EXHIBIT.—This association which numbers over seven hundred members in the

United States, Canada and Hawaii, will hold its second art exhibit in the City Art Museum of St. Louis, May 14-20, 1939, during the annual session of the American Medical Association.

Art pieces will be accepted for this art show in the following classifications: (1) oils both (a) portrait and (b) landscape; (2) water colors; (3) sculpture; (4) photographic art; (5) etchings; (6) ceramics; (7) pastels; (8) charcoal drawings; (9) bookbinding; (10) wood carving; (11) metal work (jewelry). Practically all pieces sent in will be accepted. There will be over 60 valuable prize awards.

For details of membership in this association and rules of the exhibit, kindly write to Max Thorek, M. D., secretary, 850 Irving Park Blvd., Chicago, Ill., or F. H. Redewill, M. D., president, 521-536 Flood Bldg., San Francisco, Calif.

SALMON MEMORIAL LECTURES.—Dr. Edward A. Strecker, professor of psychiatry in the University of Pennsylvania, is Salmon lecturer for 1939. He will give three addresses at the New York Academy of Medicine on April 14, 21 and 28, the theme of which will be "Beyond the Clinical Frontiers."

Abstracts of the individual lectures are as follows:

LECTURE I—THE MASSIVE RETREAT FROM REALITY.

Psychoses, psychoneuroses and other psychiatric situations are viewed from the perspective of a common factor—the evasion of reality. The individual units of reality evasion, or stereotypes, are considered in compositions and objectives. Consideration of natural, artificial and pathological reality evading mechanisms and devices. General relationships of psychiatric phenomena with the psychology of everyday life and notably with "crowd-mindedness." Discussion of factors influencing the alarming increase of "crowd-mindedness" and mob reactions.

LECTURE II—ANALYSIS OF "CROWD-MINDEDNESS."

An attempt to trace the kinships between mental and psychoneurotic symptoms and mechanisms—their miniature prototypes in everyday living—their massive display by emotionally dominated "movements" and mobs. Consideration of cultural lessons to be learned from psychopathology.

LECTURE III.

Such cultural threats as war, materialistic civilization, regimentation, narrow concepts of non-cultural physical sciences are scrutinized as emphasizing the need for mental hygiene. The feasibility of social mental hygiene and the significant rôle it must be prepared to play in any plan designed to redeem our cultures is discussed.

The Salmon Committee on Psychiatry and Mental Hygiene, appointed by the Council of the New York Academy of Medicine, has this year for the first time authorized delivery of the Salmon lectures outside the city of New York. Accordingly the material of the 1939 series, condensed into a single lecture, will be presented by Professor Strecker in Toronto under the auspices of The Toronto Academy of Medicine, May 5, 1939.

ACTA MEDICA URSS.—This new medical publication, originating in Moscow, made its appearance in 1938. It is issued by the State Publication Office of Medical Literature of the Commissariat of Public Health, under the editorial direction of Professor M. Serejski.

The purpose of the new journal, which will cover the whole field of medicine, is to present a vehicle through which the medical profession in other countries may become acquainted with current medical science in the Soviet Union. To that end articles will regularly appear in four languages, Russian, French, English and German, individual issues of the publication containing from 200 to 250 pages.

The objective of the Commissariat of Public Health to furnish in this way a medium of international exchange and to bring into closer relationship the physicians of various countries, deserves most cordial commendation. The JOURNAL extends fraternal greetings to *Acta Medica URSS*.

REPORT OF NOMINATING COMMITTEE.—In accordance with Article VI of the Constitution of The American Psychiatric Association, the Nominating Committee herewith reports the following nominations to the membership of the Association, to be acted upon

at the ninety-fifth annual meeting in Chicago, Illinois, May 8 to 12, 1939:

For President: William C. Sandy, M. D., Pennsylvania.
For President-Elect: George H. Stevenson, M. D., Ontario.
For Secretary-Treasurer: Arthur H. Ruggles, M. D., Rhode Island.
For Councillors for three years: Richard H. Hutchings, M. D., New York; Lawrence Kolb, M. D., District of Columbia; William J. Tiffany, M. D., New York; E. Armitage Baber, M. D., Ohio.
For Auditor for three years: Charles W. Castner, M. D., Texas.

C. MACFIE CAMPBELL, M. D., *Chairman,*
ROSS MCC. CHAPMAN, M. D.,
CLARENCE O. CHENEY, M. D.,
WILLIAM L. RUSSELL, M. D.

DEATH OF DR. DONALD GREGG.—Since our last issue word has come of the death of Dr. Donald Gregg, superintendent of the Channing Sanitarium, Wellesley, Mass., January 6, 1939. Dr. Gregg was a graduate of Harvard University both in arts and medicine. He had two years hospital experience in the Philippine Islands, where he served also as assistant professor of tropical medicine. Returning to the United States Dr. Gregg spent four years on the staff of the Boston Psychopathic Hospital, and in 1921 was appointed superintendent of the Channing Sanitarium, which position he held until his death.

Dr. Gregg's medical and public interests were many, including the presidency of the Boston Society of Psychiatry and Neurology, presidency of the Massachusetts Society for Mental Hygiene, membership in the Board of Trustees and secretaryship of the Walter E. Fernald School for the Feebleminded, as well as membership in numerous medical associations. He was a diplomate of the American Board of Psychiatry and Neurology.

Dr. Gregg was one of the outstanding psychiatrists in New England, and a highly esteemed citizen in his community.

AMERICAN ASSOCIATION OF PSYCHIATRIC SOCIAL WORKERS ANNUAL MEETING.—"Some Aspects of Psychiatric Social Work," published in August 1938, constitutes the conference number of

the *News Letter*,* the quarterly publication of the American Association of Psychiatric Social Workers. Here are contained in full the six papers presented at the annual meeting of the Association held in conjunction with the National Conference of Social Work in Seattle in June 1938. The first two papers deal with current developments in generic and specific aspects of social case work and with special features of psychiatric social work. The others illustrate by means of case material, the generic and specific aspects of psychiatric social work in different administrative settings, including the child guidance clinic, the psychiatric hospital and the adult psychiatric clinic. In each of these latter papers, a worker representing the particular setting attempted to demonstrate how the psychiatric social worker relates her case work to that of the psychiatrist.

The annual report for 1938 summarizes the interests and activities of special committees of the American Association of Psychiatric Social Workers; professional education for psychiatric social work, a study and formulation of commonly accepted practices of the psychiatric social worker in mental hospitals, and a study of social treatment and practices in child guidance clinics. The report emphasizes, in view of the proposed developments in state and federal health programs, the Association's interest and readiness to assist in the construction or reformulation of civil service requirements for positions in psychiatric social work. It also stresses the importance of strengthening working relationships with other professional associations having allied interests, particularly The American Psychiatric Association, as the contribution from psychiatrists is continually needed in a program geared to maintain standards and to develop further activities. During the current year, 1938-39, the program of Association activities includes further work by the professional education committee relating to formulation of a minimum curriculum for training in psychiatric social work. The mental hospitals committee is bringing to completion the interim statement of minimum standards and accepted practices in psychiatric social work departments of state hospitals and the child guidance study committee is pursuing further its study of social treatment practices in child guidance

* *News Letter* may be obtained from Miss Irma E. Mohr, Editor, 722 West 168th Street, N. Y. C. Price 50¢.

clinics. The next annual meeting of the Association will be held at the time of the meeting of the National Conference of Social Work in Buffalo, June 18 to 24, 1939.

The American Association of Psychiatric Social Workers had its beginning in 1920 as a small informal group of social workers living in or near Boston. It is a growing organization, with a present membership of 520 workers, located in thirty-three states, Hawaii, Canada, England and Scotland. Membership is basically homogeneous. In addition to the general education and formal professional training required for junior members, composing approximately twenty per cent, the active members must have had from one to four years of clinic or hospital experience (depending upon the extent and type of training) in association with a psychiatric staff.

The Association, by virtue of its membership requirements, embodies relatively high professional standards. It has played a part in the raising of standards of psychiatric social service in psychiatric clinics and hospitals and in the formulation of standards of training. It has made a contribution to allied fields of social work where some of its members are actively employed and where there is increasing appreciation of the psychiatric implications in all phases of social case work. Employers also reflect a growing recognition of the value of experience gained in psychiatric hospitals and clinics. Present figures show that approximately 50 per cent of the Association's members are working in the field of psychiatry, 25 per cent are concerned with mental hygiene education, and approximately 25 per cent are practising in various social case work agencies. There is a demand by schools of social work for more psychiatric training centers and increasing desire among students and social workers for experience in the psychiatric field.

Officers for 1938-1939 are: President, Mrs. Katharine M. Wickman, Babies Hospital, New York; Vice-President, Miss Lois M. French, New Jersey State Teachers College, Newark; Secretary, Miss Leona M. Hambrecht, N. Y. State Psychiatric Hospital, New York; Treasurer, Miss Sylvia B. Astro, Bureau of Child Guidance, Board of Education, New York.

LEONA HAMBRECHT.

SIXTH BIENNIAL CONGRESS OF THE INTERNATIONAL HOSPITAL ASSOCIATION.—The Sixth Biennial Congress of the International Hospital Association will be held in Toronto, Canada, September 19 to 23, 1939. The headquarters for the Congress will be the Royal York Hotel.

This meeting of national and international hospital men and physicians affords an unusual opportunity for the presentation of able papers and discussions on hospital organization and administration, and facilities for the care and treatment of the sick.

In contemplation of an international hospital association, hospital representatives from various countries first assembled in Atlantic City, New Jersey, in 1929. The meeting of formal organization was held in Vienna two years later. Since 1931, biennial meetings have been held in Belgium, Italy and France. The Association has now grown to sturdy proportions, hence the 1939 meeting in Toronto, Canada, should prove outstanding.

The high hopes of the Association are ably expressed by Dr. W. Alter, one of its founders, in his editorial in the January number of *Nosokomeion* (X: 21-22), the official journal of the Association:

> The fact that the first steps toward the founding of the I. H. A. had been made in America gave rise to hopes that the Association would form a living bridge between the glorious traditions of the hospitals of the old world and the no less glorious modernity of the hospitals of the new. This hope has alas! been only partially fulfilled. It is the deep and sincere wish of all members of the I. H. A. that the 1939 Congress which leads the Association back to the land of its cradle, shall consolidate there once and for all the grand, strong, energetic interest which it so urgently needs from America, and would so greatly value. The perfect hospital of the future, which is the ideal we are all striving for, requires a wealth of experience from every country in the world for its shaping and realisation. And America is, by the very magnificence of its own hospital achievement, called upon to furnish some of the cornerstones for that edifice.
>
> The I. H. A. looks forward to the Toronto Congress not only in the hope that the meeting will lift some of the shadows that have darkened its work, but in the strong belief that great and important progress will be made in all fields of hospital work and a valuable forward step will be taken towards developing the hospital into a community institution, eager to serve with the best possible means and with selfless devotion the greatest purpose of humanity, the perfection of its mental and bodily health.

Dr. Malcolm T. MacEachern, president of the International Hospital Association, emphasizes its common objective—the care of the patient—(*Nosokomeion*, X: 24-26):

> Let that service be our common bond! Not as different people, different hospitals, or different nations, but as one huge unit, one huge army, let us fight disease, all working toward our greatest objective—the saving of human lives—and in our associations thereby developing that friendship, that comradeship, in which there is strength, encouragement and stimulus to do our work better and still better!

The biennial Congresses of the Association are supplemented in the intervening years by instructive Study Tours conducted in the various countries. Research is fostered and results reported through the Association's forty Study Committees. These Study Committees cover all details of hospital organization and management, and all the activities of professional service are fully represented. For a full outline of the program of the Toronto meeting of the Association, consult the official journal of the Association. Your personal interests are sure to be touched upon. Should you be especially interested in the Study Committee in Neurology, address Dr. F. H. Lewy, University Hospital, 3400 Spruce Street, Philadelphia, Pennsylvania. Again, should you be especially interested in the Study Committee in Psychiatry, address Dr. Thos. J. Heldt, Henry Ford Hospital, Detroit.

The officers of the I. H. A. extend to you a cordial invitation to attend the Association's Toronto meeting.

THOS. J. HELDT.

PSYCHOSOMATIC MEDICINE.—The first issue of this new journal has appeared bearing the date January 1939. The purpose of this publication, which has been initiated with the assistance of the Josiah Macy Jr. Foundation, is to promote studies dealing with the organism as a whole, bridging the gap between somatic and psychological medicine.

The material to be published the editors explain, "will deal primarily with phenomena observed concurrently from somatic and psychic angles rather than from either one alone. The scope therefore will include appropriate experimental studies of animal and human behavior, and well controlled clinical studies of both children and adults."

Psychosomatic Medicine will appear quarterly under the sponsorship of The Committee on Problems of Neurotic Behavior, Division of Anthropology and Psychology, National Research Council, Washington, D. C. The managing editor is Dr. Flanders Dunbar, assisted by a board of editors consisting of Drs. Franz Alexander, Dana W. Atchley, Stanley Cobb, Hallowell Davis, Clark L. Hull, Howard S. Liddell, Grover F. Powers, Theodore P. Wolfe, and an advisory board of thirty-eight physicians representing various fields of medicine.

The first issue includes a contribution by Franz Alexander on "The Psychological Aspects of Medicine", a review of recent contributions on the hypothalamus by Roy R. Grinker, W. R. Ingram and S. W. Ransom and a symposium on hypertension to which eleven authors contributed.

It is proposed that longer studies will be published as monograph supplements from time to time.

Book Reviews.

RESEARCH IN MENTAL HOSPITALS. By *The National Committee for Mental Hygiene*, N. Y., 1938. A survey and tentative appraisal of research activities, facilities and possibilities in state hospitals and other tax-supported institutions for the mentally ill and defective in the United States.

From a considerable mass of data accumulated by the field representatives of the National Committee, Dr. Charles P. Fitzpatrick and Dr. Winfred Overholser, with assistance from others, an attempt has been made, in this paper-bound volume of 151 pages, to present a general picture of psychiatric research *in tax-supported institutions*. The book is not a critical appraisal of actual research projects and programs. It contains no bibliography. It does not attempt to present the findings of individual investigators nor to evaluate the psychiatric significance of their work. As Dr. Hincks states in the foreword, it is hoped that "it will prove of interest and value to hospital executives, hospital trustees, legislatures, budget-making bodies and other governmental authorities, and to Foundations and Funds who are turning their attention and financial support increasingly to this promising field of medical science." To the reviewer it seems probable that the actual detailed information about specific institutions which is in the files of the Committee may prove more potent for such purposes than the rather pallid abstract summarization presented here.

The bulk of this book is based on replies to a questionnaire, which is perhaps inevitable in such a survey, but does invite criticism. An example may illustrate one of the less meaningful results so elaborately derived. One may learn on page 110 that "for the country as a whole the median hospital has 29.9 per cent of its medical staff interested in research." This statistic need not, however, be taken very seriously. It is probably a typographical error anyway, since table 29 and graph 9 give 22.9 per cent. Those actually familiar with psychiatric research activities will react with some surprise to the evidence from this table and graph that the medical staffs of state hospitals in the East South Central States (Alabama, Kentucky, Mississippi and Tennessee) have research interests statistically equivalent (26 per cent) to those in the New England states, and somewhat greater than those in New York, New Jersey and Pennsylvania (19.6 per cent). This curious result becomes more readily understandable when one discovers on page 123 that the Worcester State Hospital, the Boston State Hospital and the Boston Psychopathic Hospital are not included in the statistical evaluation of New England. The research activities in these institutions and a number of other centers, are discussed in another portion of the book.

The questionnaire gets to a firmer foundation in other items considered in evaluating the possibilities of research in the state hospitals: e.g., the ratios of patient load to medical staff, and nursing staff, the facilities for teaching internes and junior physicians, relationships with medical schools and universities, facilities for library and laboratory work, etc. No data or ratings for individual institutions in these respects are published here—in accordance apparently with the general policy of anonymity. Median figures are given, characteristic for each region of the country. From the data and appendices given, it would seem possible to apply the rating scheme to any specific institution on which one had the requisite detailed information, and thereby compare it with the regional median and the median of the country at large.

Part III presents thumb-nail sketches of twenty research centers which operate in tax-supported institutions, although the research activities in a number of these centers are in large portion supported by foundations, individuals and other private agencies.

Part II and Part I are devoted to the interpretation of findings and a summary.

The keynote of the volume is expressed in this sentence: "The survey has furnished tangible evidence to indicate that a creditable beginning has been made in the development of research in public institutions, that there is a genuine interest on the part of hospital superintendents in the extension of investigatory work, that many worthwhile developments might be fostered with modest encouragement and financial support, and that the time is ripe for the inclusion of psychiatric research as an integral part of public policy in a number of states that hitherto have devoted little attention to this matter."

The aim of this survey is altogether praiseworthy, there is evidence that an abundance of valuable information is on file with the Committee, but the reviewer feels obliged to state that to him the present book seems hastily and carelessly prepared, and ineffectively presented. It is earnestly to be hoped that the extensive, important and detailed survey of psychiatric research being conducted by the National Committee should not be judged from the inadequate fragment presented in this book.

JOHN C. WHITEHORN, M. D.,
Washington University, St. Louis.

CONCEPTS AND PROBLEMS OF PSYCHOTHERAPY. By *Leland E. Hinsie.* (New York: Columbia University Press, 1937.)

Professor Hinsie states "The purpose of this book is to indicate the general conceptions that prevail with respect to the structure and functions of the mind, and to show, as well as possible, what influences these conceptions have had upon the problems of psychotherapy."

The introduction is devoted to a general discussion of the present status of the theory and practices of psychiatry. Dr. Hinsie's familiarity with the modern methods under discussion free his views from bias and result in a

fair, reasonable evaluation of our present knowledge. The introductory chapter alone would give the book a place in psychiatric literature.

The major portion of the book is divided into three chapters devoted to a discussion of psychoanalysis, psychobiology, and individual psychology and analytical psychology respectively.

The chapter devoted to psychoanalysis presents the fundamental Freudian concepts in a logical, simple style, free of the excess defenses that characterize so many similar presentations now present in our libraries. The section on choosing patients for analysis will be useful to physicians in other fields of medicine who have patients they wish to refer for this treatment.

Chapter 3 discusses psychobiology. The presentation is brief, and rather superficial, but it conveys the general attitude of the Meyerian group and has the unique distinction of presenting the method in simple, readily understood terms. The open-mindedness and general usefulness of the psychobiologic approach is stressed.

The works of Adler and Jung are presented briefly, but concisely. The more generally accepted and useful portions of their doctrines are discussed in some detail.

Chapter 5 is entitled "Statistical Evaluation" and is written by Carney Landis. His fair handling of the available data compels the reader to the conclusion that the analytic literature contains a paucity of statistical material.

The sixth and final chapter, "Conclusions," is largely a plea for, and defense of, statistical method and adds little of value to the book.

The bibliography is excellent.

This volume is a brief, practical, unprejudiced presentation of the principal present day concepts of psychiatry, and is a book which should be in the library of every teacher and student of psychiatry.

F. G. E.

PSEUDOCYESIS. By *George Davis Bivin, Ph. D., and M. Pauline Klinger, M. A.* (Bloomington, Indiana: The Principia Press, Inc., 1937.)

In this book Bivin and Klinger present an intensive analysis and summary of 444 cases of so-called pseudocyesis, which have been collected from accessible records throughout the world. With care as to details and with evident exactitude these cases are discussed under the headings of symptomatology, diagnosis, etiology and treatment. No new hypotheses are advanced but a historical survey of etiological theories and a very comprehensive discussion of symptoms in chronological order comprise the major content of the book.

It is an impartial and illuminating description of the condition, a monograph compiling an enormous amount of material in an understandable, readable style. As a reference book on the subject it is undoubtedly of value.

CLARA L. HOYE, M. D.,
St. Elizabeths Hospital, Washington, D. C.

PSYCHOTHERAPY. By *Paul Schilder, M.D., Ph.D.* (New York: W. W. Norton and Co., Inc., 1938.)

It would be of value to psychiatry and to medicine as a whole, if those who advance therapeutic systems would from time to time publish the criteria of proof upon which they rest their case. Ordinarily speaking, phenomena are firmly established as facts in medicine by experimentation. If that is not possible, control studies are done in which untreated cases are compared with similar groups of individuals who are treated. If a theory is advanced as to genesis or causation, then statistical studies are made which, while they are not usually conclusive, give some validity to the statements made. Thus, if the solitary child is more predisposed, let us say, to the anxiety neuroses than the child of the large family, the situation may be studied, first as to the relative incidence of the solitary child in the anxiety states; then solitary children might be studied for the incidence of the neuroses, and these results compared with the relative incidence of solitary children in the total population, etc.

No matter how satisfying the statements may seem to the authors thereof, no matter how ingenious the line of reason or argument may be, all the self-satisfaction in the world and all the ingenuity prove nothing. If one has a working hypothesis upon which he operates, which cannot be proven or disproven, then the fact that there is such a working hypothesis should be definitely stated, but no wide-reaching claim made as to the truth or validity of the hypothesis until such time as the hypothesis is proven or disproven.

No such approach appears in this book by Dr. Schilder. That essential humility before the unknown and unprovable, which should govern scientific discussion and statement, appears conspicuously absent in this volume. In this the author does not differ from the approach made by psychoanalysis as a whole, although latterly the idea that statements have to be proven seems to be seeping into the scientific consciousness of the workers in this field. All that is stated in this book may be true, but nowhere in the volume does there appear any proof and none of the things to which Dr. Schilder refers has actually been proven.

A typical statement is this (p. 24): "The psychotherapeutic relation between the physician and his patient thus becomes a mirror of possible social relations between two human beings, in which the physician is no less affected than the patient himself." Clearly, this is exaggeration and is mainly literary. Murder, sexual intercourse, financial relationships, the master-slave and slave-master relationships—none of these is present. Moreover, if the physician were really as affected as the patient, he could not take care of more than one case; he could not see patients from nine in the morning until five in the afternoon, with an hour off for lunch, and remain sane or well. Thus, the statement is partly true, but in its sweeping totality it is not true.

Similarly the author states (p. 28): "The whole secretion in the gastro-intestinal tract is dependent upon psychic influences." It is modified by

psychic influences, and unless the whole world is made psychic, including acids, the statement is obviously incorrect, as it neglects the other types of influence. This is not a puristic criticism; it is a criticism of the tone of the entire book.

The chapter on the symptomatology of organic disease has many interesting, almost fascinating statements which, however, to the reviewer are interesting and fascinating because they assert much and prove little or nothing. The chapter is one mass of statements which may or may not be true. It is possible that proof is adduced elsewhere, but a little excursion into the references reveals merely another set of assertions. Here is a typical statement (p. 32): "When a person suffers by organic disease, there is no need for further punishment, and he may be relieved from his feeling of guilt." If the feeling of guilt, however, is manifested by anxiety, then the sufferers from organic diseases are not relieved of guilt, since one frequently sees very anxious people with organic disease, and furthermore sees anxiety states, hysteria and other conditions as part of the picture of organic disease.

A whole range of questions is begged in the following statements (p. 41): "Paralysis and weakness, as well as fatigue, express the unwillingness to act. . . . . Epileptic fits organically determined are very often connected with the expression of severe aggressive and sadistic tendencies." But severe aggressive and sadistic tendencies may exist without epilepsy, may exist in a hundred and one different conditions, as has been proven to their own satisfaction by the psychoanalysts themselves, so that the matter finally comes down to this, that severe aggressive and sadistic tendencies are found everywhere amongst human beings.

The following beautiful and far-reaching statement is also made (p. 83): "It is trite to say that every sick person needs psychological help. We can be more specific since there is the tendency of the organic case to forget about his organic disease or to minimize it. We have to lead him back in this respect to reality. It is indispensable in the treatment of the organic case to bring the patient to an appreciation of the reality of the disease. Non-appreciation of the disease and the experience of health are in many of these cases dangerous. . . . ."

One is left baffled by these statements. There is a modicum of truth in them, but there are any number of organic diseases in which knowledge of "reality" has nothing to do with recovery. A pneumonic exudate will clear up irrespective of the knowledge of the patient. The value of insulin in the acidosis of diabetic coma has nothing to do with the appreciation of the illness. The paretic is helped by hyperthermia, willy-nilly, etc.

One could go through the book from page to page, finding this type of loose statement (p. 309): "There is no doubt that in schizophrenia the patient goes back to a more primitive mode of existence." In whose mind is there no doubt? The bulk of psychiatrists would doubt this very strongly. There is regression if by this is meant deterioration, but the schizophrenic

in no wise resembles primitive man or the healthy child. Much more has been accomplished by pharmacology than by psychoanalysis in schizophrenia. Much more is being accomplished by physiological means combined with very simple psychological approaches than by all the subtleties that fill the books from Freud to Schilder. Insofar as results give clues to genesis, it would be far wiser to say that there is a pharmacological deficiency than to make any psychological conclusions about schizophrenia at the present time.

The section concerning symbols expresses the well known notions that (p. 128) "everything which is pointed, which protrudes, which is like a stick, is used as a symbolization for the male sex organs. Everything which is formed like a box and which has an opening can be used as a symbol for the female sex organs," etc. Obviously one can prove anything one wants to with symbols of this type, since as Southard once said, "Everything is either somewhat straight or somewhat round." I wonder why the analysts have thus far neglected the human nose which is the perfect symbol for hermaphroditism since it is both pointed and contains openings which lead into a box. A rich field has been hideously neglected.

Those who believe in analysis and its various modifications will be delighted with this book; those who do not will have a good time reacting hostilely. A disproportionate space is given to the Freudian ideas and methods. Adler, Jung and Adolf Meyer are very cavalierly disposed of in a page or two or three. Most of the book is Schilder modifying Freud.

The book is worth the reading and worth the owning. There are passages of excellent insight and the author's wide experience shows up continually. It may be entirely the fault of the reviewer that his critical judgment and his objection to wide, all-inclusive statements without proof are outraged on every page.

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PSYCHOLOGICAL FACTORS IN MARITAL HAPPINESS. By *L. M. Terman.* (New York: McGraw-Hill Book Company, 1938.)

This is a report of investigations on 792 married couples regarding several hundred possible factors correlated with measures of "marital satisfaction." It is very clear as to the methods employed in the investigations, the criteria adopted, the statistical devices by which the data were systematized, etc. The conclusions drawn cannot be summarized in a brief note, save that they point to the statement that "genuine research on the causes of marital unhappiness has barely begun" (p. 377). The reader will discover much interesting data in the report, some of which may assist in dispelling a few popular misconceptions. He is likely to feel, however, that the book, through its title, promised more than it gave.

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GENERAL PSYCHOLOGY FROM THE PERSONALISTIC STANDPOINT. By *William Stern*. Translated by Howard Davis Spoerl. (New York: The Macmillan Company, 1938.)

This presentation of Stern's system of psychology commands the serious attention of the scientific world. Resting, as it does, upon a lifetime of psychological research in almost every field; upon a consistent plan of tentative systematic formulation and constant revision; and upon the firm background of the author's metaphysical and ethical contributions, it constitutes one of the most formidable books in modern psychological literature. Furthermore, although Stern's basic concepts have been familiarly used by him for many years, the present publication is indeed quite up-to-date; for it takes its place with those other bold present-day attempts to demonstrate that psychology can and must regard the human individual as its object of study. As Allport in his excellent critical description of this book says (*Character and Personality*, 1937), "In stressing the person, the author with remarkable directness calls psychologists back from their esoteric bush-beating and confronts them with their forgotten man."

Part I deals mainly with the presuppositions and basic concepts of psychology as a branch of personalistics. Subsequent parts review the more significant data of psychology under the traditional headings Sense-Perception, Memory, Thought and Imagination, Striving, Feeling, at the same time interpreting all concepts and data from the personalistic standpoint. The interpretation is consistent throughout, even though at times the variety of phenomena discussed may make it difficult for the reader to be always conscious of that consistency.

Further comment is unnecessary, unless the reviewer should launch into a description or critical evaluation of Stern's point of view. Several descriptive summaries of that point of view have already appeared in psychological journals. Adequate critiques have yet to appear. It is sufficient at this time to remark that Stern's account speaks for itself. His contribution has obviously influenced many of the leading writers in the field of Personality (G. W. Allport, for example). The permanent value of his contribution may lie in the stimulation it gives to the formulation of a Psychology of the Person that will actually and systematically portray the nature and uniqueness of His development, rather than being content with an insistent description of Him *as* unique.

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OUT OF MY LIFE AND WORK. By *August Forel*. Translated by Bernard Miall. (New York: W. W. Norton & Co., 1937.)

Autobiography is always an illuminating and interesting type of literature; this is particularly true when, as in the present case, the subject is a great figure who by training has learned to look with reasonable objectivity at his motives and accomplishments.

There is little need to rehearse the biographical data of August Forel; his significance is well known to all psychiatrists. As an early contributor to the field of neuroanatomy, as a pioneer in hypnotism and in occupational therapy, as the predecessor of the great Bleuler at Burghölzli, as an enthusiastic campaigner for total abstinence, and as a profound student of that very interesting insect, the ant, his energy, his wide interests, his broad social vision, and his psychiatric and neurological pioneering make his place in history secure.

This autobiography sketches intimately and engrossingly the development of a shy, isolated, unhappy boy, finding companionship only in his beloved wasps and ants, into the serious student and teacher and social reformer that Forel became. Of particular interest to the psychiatric reader in his account of the days at Burghölzli, of his successful contests with the cantonal authorities who hampered the professional development of the hospital (even Switzerland is not immune!) and of his efforts to bring Burghölzli to the standing it has now long enjoyed.

Big-hearted, studious, of indomitable will, strong convictions, and rigid uprightness, ever seeking for new knowledge, the world lost a great figure in the death of August Forel. Anyone interested in social progress or in a broad view of psychiatry can read this book with pleasure and with much profit.

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THE 1938 YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND ENDOCRINOLOGY. Edited by *Hans H. Reese, M. D.* (Neurology), *Harry A. Paskind, M. D., Ph. D.* (Psychiatry), *Elmer L. Sevringhaus, M. D.* (Endocrinology). (Chicago: The Year Book Publishers. 1939.)

A periodic inventory of progress, indispensable in any field of science, is peculiarly necessary in the rapidly expanding domain of medicine. Such an inventory the series of eleven year books, representing the major departments of medicine, supplies. They belong in every medical library, and both general practitioner and specialist will require at least some of the annual volumes for ready reference.

The 1938 Year Book of Neurology, Psychiatry and Endocrinology presents reviews of 520 contributions selected from 104 periodicals published in the United States and 15 other countries.

It has occasionally happened that reviews of articles of questionable value, appearing in leading medical journals, have found their way into the Year Book. This contingency the editors have striven to avoid, and a notable feature is the inclusion of numerous editorial comments throughout the text for the guidance of the reader as he surveys the material presented. New offerings in the way of observations and theories are thus made available, but with footnotes which call attention to conclusions which may be based on inadequate evidence or require confirmation.

Especially noteworthy is the increasing space devoted to questions of treatment, reflecting intense activity in each of the three departments represented in the Year Book. The reader will find epitomized the newer therapeutic procedures in encephalitis, poliomyelitis, sulfanilimide therapy in the meningitides, the use of new drugs in migraine and other allergic conditions, hormone treatment of menopausal symptoms, specific and non-specific therapy of paresis and tabes, the use of the irradiated sterol "A. T. 10" in tetany, the specific treatment of pituitary disorders, a fair representation of the multitude of contributions on the insulin and metrazol treatment of schizophrenia and other mental disorders, in which zeal and optimism are wholesomely balanced by reports of pathologic changes in the central nervous system of patients dying after shock treatment.

The Year Book is provided with both author and subject indexes and contains 131 illustrations. Within its 776 pages is condensed the outstanding work of the year.

C. B .F.

In Memoriam.

HUGH TALBOT PATRICK.

1860-1939.

Hugh Talbot Patrick, eminent neurologist, was born at New Philadelphia, Ohio, May 11, 1860. After his university education he graduated in medicine from the Bellevue Hospital Medical College (New York University) in 1884. After his internship he began the practice of medicine in Chicago in 1886.

From 1891 to 1894 he did graduate work in nervous and mental diseases at the principal medical centers in Germany, France and England, maintaining ever since friendly and professional relations with the leaders of English and Continental neurology. Dr. Patrick's teaching activities began in 1895 when he was appointed instructor in the medical school of Northwestern University. Three years later he became associate professor, and in 1902 professor of nervous and mental diseases. He served also as attending neurologist of the Wesley Memorial Hospital, the Passavant Memorial Hospital, the People's and Henrotin Memorial Hospitals, St. Anthony's Hospital and the Illinois Charitable Eye and Ear Infirmary.

Dr. Patrick was a founder of the Chicago Neurological Society, its first secretary and twice its president. He was a member of the American Neurological Association, twice its vice-president and president in 1907; of the Institute of Medicine of Chicago, of which he was once president; of the American Medical Association, and secretary and chairman of its section on nervous and mental diseases; of the Chicago Medical Society of which he was a trustee; of the Mississippi Valley Medical Association of which he was once president; of the Illinois State Medical Society, Chicago Pathological Society, Chicago Society of Internal Medicine, the Central Neuropsychiatric Association, Chicago Academy of Medicine, Association for Research in Nervous and Mental Diseases, and foreign corresponding member of the *Societe de Neurologie de Paris.*

He was the author of numerous scientific treatises, and collaborator in the authorship of several books. He was instrumental in founding the Archives of Neurology and Psychiatry, was its chief editor, and the first to receive the honor of a dedicate number of the Archives of Neurology and Psychiatry in 1936. He was a member of the editorial board of the Journal of Nervous and Mental Diseases. For many years he was the editor of the Year Book of Nervous and Mental Diseases.

Despite his age he served his country as contract surgeon during the World War.

He was an honorary member of Alpha Omega Alpha.

He was the recipient of a distinguished honor when he was elected by the Republic of France to membership in the Legion of Honor with the rank of Chevalier.

He was a member of the Physicians Club, the University Club, the Santa Barbara Club, Glen View Golf Club, etc.

He was married April 28, 1896, to Fannie E. (deceased), daughter of Joseph E. Gary, for over 40 years judge of the superior court of Chicago. They had three children, Talbot, Catherine, and Elizabeth Patrick (deceased).

When the honorary degree of Doctor of Science was conferred upon him in 1927 by Northwestern University the *Causa Honoris* read as follows: Hugh Talbot Patrick, distinguished neurologist of international fame; profound student and stimulating teacher; clinical investigator of great achievement; inspirer of youth; *Chevalier de la Legion de l'Honneur* of France; servant of his country in time of need; Emeritus Professor in this University and a Counsellor of Medicine; a learned, kindly physician.

LEWIS J. POLLOCK, M. D.